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The E. C. Stirling Lectures.[1]

LECTURE II.

FACTORS IN THE PROGNOSIS OF CARDIAC DISEASE.

By A. W. HOLMES À COURT, M.D., F.R.C.P.,
Sydney.

SYMPTOMS are universally available: they are the voice of Nature: signs, by which I mean more artificial and refined methods of scrutiny, the stethoscope, the microscope etc. are not always within the power of every man, and with all their help are additions not substitutes. (John Brown: Introduction to "*Horæ Subsecivæ.*")

Estimation of prognosis in cases of illness is probably the most difficult and frequently the most uncertain part of our professional work. It is proposed to place before you some general principles applicable in particular to estimation of the outlook in cardiac disease and to stress the importance of clinical observation.

In considering the heart and circulatory function it would appear first necessary to determine by what evidence cardiac failure may be recognized clinically, particularly in its earliest stages, and in what type of patient the failure of the cardiac mechanism may be foreseen. In order to reach a clear understanding of this problem it is desirable to formulate some means by which the functional efficiency or otherwise of the circulatory mechanism can be adjudged.

It is obvious that this inquiry is primarily concerned with function rather than structural or pathological defect, except in so far as structural alteration determines impairment of function. In other words, it is the ability of the heart to carry

[1] Delivered at Adelaide in May, 1938.

out its work which is the important question, rather than the presence or absence of signs of changes in the valve cusps and of abnormal cardiac sounds. Let us therefore endeavour in the first place to arrive at a method of investigating the early evidence of disturbance of function. To do so it is obviously necessary to define what constitutes healthy or normal cardiac function, or by what means we can say with certainty that the heart of a patient is normal in its ability to carry on the circulation. As this inquiry is concerned with function, it is apparent that departure from normal is likely to be made manifest primarily by subjective symptoms rather than by physical signs. Hence it is that at the present time, when discussing cardiac failure, the older nomenclature of cardiac disease, in which classification according to structural defects formed the basis, has been largely replaced by a new terminology in which disorders of function or working capacity are the foundation. Thus reference is made to cardiac breathlessness, failure with congestion, cardiac enlargement, effort syndrome, hypertension, disorders of rhythm (for example, auricular fibrillation), cardiac ischæmia and coronary occlusion, pulmonary œdema, and so on. In discussing cardiac failure, such terms as rheumatic carditis, subacute bacterial endocarditis, coronary sclerosis, syphilitic aortitis and the like, are used to form the basis of pathological classification rather than the older terms, mitral stenosis, aortic regurgitation and similar forms, in which alteration in structure involving the valves is the foundation and ætiological factors are not indicated.

The term "failure of compensation" has been generally discarded in speaking of cardiac breakdown.

The central problem for consideration in the present inquiry is the failure of the heart to accomplish its work in lesser or greater degree, irrespective of its structural peculiarities. In practically all cases of cardiac failure breathlessness is the initial symptom of breakdown of the circulatory mechanism, varying in degree from shortness of breath on exertion, more pronounced than the breathlessness of the healthy person upon severe effort, to constant distress of breathing when the body is at complete rest. It is therefore apparent that the first evidence of impairment of circulatory function will be manifest as a restriction of the field of cardiac response to effort; some form of physical exertion previously within the range of the powers of the individual becomes "conscious effort" associated with breathlessness; in short, the cardiac reserve has been encroached upon and there is found to be a diminished tolerance of exercise. Hence it is that the first evidence of cardiac disorder is to be sought from the symptoms of the patient, although judicious questioning may be necessary to explore the limitation of cardiac reserve. In order that some criterion may be established for such investigation, exercises tests have been devised to test out cardiac efficiency. (See Lewis, "Diseases of the Heart".)

Tests of Cardiac Efficiency.

Simple Test.

(i) The subject walks briskly up forty steps, one at a time, and is examined at once; or (ii) the patient takes twenty hops on the right foot and twenty on the left, raising the shoulders about six inches each time; or (iii) the patient steps on and off an eighteen-inch chair twenty times.

A healthy young subject will show no obvious distress from such exercises, the pulse rising no more than 10 or 20 per minute, the original rate being resumed after a minute or a little more.

Should there be a well-marked reaction to such a simple test, provided the breathlessness is definitely of cardiac origin, the patient belongs to the class in which cardiac reserve is largely exhausted but not as yet necessarily to the point of breakdown of circulation.

Strenuous or Severe Test.

This test aims at producing breathlessness, and consists in the patient's lifting a weight of 4·5 to 9·0 kilograms (ten to twenty pounds) from the floor, the arms flexing and then extending above the head at the rate of one lift in two seconds.

A healthy young man of sedentary habit can do this 30 to 60 times before becoming breathless and wishing to stop. Unhealthy persons in whom the reserve is encroached on will be distressed far earlier, and the effort should be stopped at the first evidence of embarrassment. Such a test should obviously be employed only in cases in which response to the simple test is satisfactory.

Accepting the thesis that breathlessness on exertion is the usual primary symptom of failure of the cardiac mechanism, we are now in a position to discuss the clinical aspects of cardiac failure in its particular manifestations, of which undue fatigue, rapid or disordered heart action, and pain are other common symptoms, and of which venous congestion, enlargement of the heart and dropsy are the prominent physical signs.

Cardiac Failure with Congestion.

The first signs of breakdown of the circulatory mechanism are often to be found in the systemic veins. Observation of the distension of the great veins of the neck in a patient lying supine with the head raised slightly on an ordinary pillow will frequently yield valuable information of the presence of venous congestion. Normally the external jugular veins are seen to be full and distended in their lower parts, the distension ending at a point approximately level with the *manubrium sterni*, the level depending on atmospheric pressure and varying with the position of the body. As venous pressure is increased the vein becomes distended throughout its length, ultimately remaining swollen, though the head and neck are in the vertical position.

In its later stages evidence of venous congestion in the viscera becomes manifest by congestion and œdema of lungs, hepatic enlargement, albuminuria and hydrostatic œdema.

Congestive cardiac failure is to be regarded as one of the gravest signs of cardiac disease. The prognosis depends chiefly upon consideration of the ætiological factors which have caused the heart to become inefficient in maintaining circulation. It is obvious that the heart may become temporarily inefficient as a result of toxæmia that has resulted from an acute infective disease, for example, typhoid fever, influenzal bronchopneumonia, tonsillar or dental infection, septic processes and the like, the functional efficiency of the heart muscle improving when the ætiological factors have been removed. Congestive failure associated with long-standing rheumatic carditis or due to failure of heart muscle, determined by degenerative changes, such as those associated with arteriosclerosis, is obviously of most serious prognostic import, the immediate outlook being modified by the response to treatment according to accepted principles.

Principles of Treatment of Congestive Cardiac Failure.

Ætiological Indications.

Where there is an immediate causal factor that can be eliminated, its removal is indicated. Cases of this group include toxic absorption from foci of infection, thyreotoxic goitre, the anæmias and syphilis. Extraneous poisons, alcohol, tobacco *et cetera* frequently call for consideration as accessory factors.

Pathological Indications.

It is to be recognized that increased resistance in the peripheral circulation is an important factor in increasing the burden of work thrown upon the heart. Venesection is a useful though neglected therapeutic measure, particularly applicable to acute congestive failure with cyanosis, especially when acute œdema of the lung is imminent, which has failed to respond to the injection of morphine in a dose of 0·015 gramme (one-quarter of a grain) hypodermically and oxygen inhalation. The well-known *pilula diuretica* is of value, particularly in chronic cases. Of the newer diuretics "Salyrgan", injected intramuscularly, is the most effective. It is apparent that removal of gross œdema and serous effusions is thus indicated for purely mechanical reasons. This objective may be attained by measures calculated directly to relieve the strain of work thrown upon the circulatory mechanism, rest, sleep, diet and general therapeutic measures, including the administration of digitalis in the cases in which it is indicated. In other instances specific measures must be used to produce diuresis.

Clinical Indications.

The particular aim is to procure relief of symptoms. Sleep is usually best procured by barbiturates, 0·3 gramme (five grains) of "Veronal", 0·45 to 0·6 gramme (seven and a half to ten grains) of "Medinal" *et cetera*, or, when associated with pain or severe cardiac distress, by morphine given hypodermically.

Digitalis in Cardiac Disease.

Although it is universally accepted that it is in cases of congestive failure associated with irregular action of the heart due to auricular fibrillation that the dramatic effects of treatment with digitalis are observed, there is tardy recognition of the fact that adequate dosage is essential. Relatively large doses are required to produce a full digitalis effect, and a "maintenance" dose is usually of the order of about 1·2 to 1·8 cubic centimetres (20 to 30 minims) of the tincture a day. Of the value of digitalis in cardiac failure, with regular heart action, the evidence is less convincing, although it is generally agreed that in certain instances definitely beneficial results are obtained. The recent addition of "Digoxin", which may be employed by intravenous injection where an immediate digitalis effect is demanded, marks a valuable therapeutic advance. "Digilanid", recently introduced for intravenous use, is also effective, and has the added advantage of being supplied in an ampoule in injectable form, the addition of saline solution being unnecessary.

Cardiac Failure of Anginal Type.

Cardiac Pain and its Significance in Prognosis.

It is axiomatic that when an organ is performing its functions normally, with the body at rest, subjective evidence of its activity does not rise to the level of consciousness. Thus when the cardiac mechanism is unduly stressed or the circulation is beginning to fail, the patient may become conscious of the action of his heart. The conscious impression may be of undue rapidity or alteration of rhythm, or a sense of dull pain or weight may be complained of in the region of the heart, or the cardiac impulse may be described as unduly "heavy" or "throbbing". In cases associated with gross cardiac disease severe pain of anginal character may arise. For the clinician it is of importance to determine the meaning of such subjective symptoms and to assess their prognostic significance in each individual instance.

Cardiac pain of anginal type is now commonly regarded as essentially due to ischæmia of heart muscle, which may be relative or absolute: relative when the heart muscle is deprived of adequate blood supply to meet the requirements of increased effort, as in angina proper (Heberden's angina), in which a temporary inadequacy of coronary supply is regarded as the basis; absolute when a coronary artery or one of its branches becomes occluded—coronary thrombosis or occlusion.

The production of cardiac pain by ordinary effort is therefore to be regarded as a certain sign of cardiac disease. The malady described by Heberden as *angina pectoris* is the commonest condition in which this type of pain is produced on effort. This is a disease of middle-aged or elderly men, usually well fed and of sedentary habit. General arterial disease is frequent, and hypertension is found in a considerable number of cases. The production of cardiac pain on effort in such individuals is therefore to be looked upon as a serious symptom. When pain of this kind occurs in resting subjects it is

almost invariably an indication of grave disease and imminent danger.

The importance of the symptoms breathlessness and pain on effort has as yet failed to receive due recognition from the viewpoint of prognosis, notably in the assessment of risk in life insurance practice, where undue significance is still attached to the presence or absence of murmurs and the blood pressure readings, to the neglect of the more clamant warnings of encroachment on the cardiac reserve.

The condition described as effort syndrome, athlete's heart or soldier's heart, in which breathlessness and subjective cardiac symptoms, often associated with rapid heart action, cardiac murmurs and elevation of blood pressure during exercise occurs, demands close scrutiny when assessment of the relation of cardiac symptoms to prognosis is undertaken. In this condition there is a disorder of the heart in the direction of overaction, in the genesis of which nervous influences, usually associated with low-grade chronic infection, are the important factors. Although overaction and rapidity of the heart are conspicuous, the absence of cardiac enlargement and evidence of other structural changes is significant in arriving at a diagnosis.

Having considered the broad principles involved in the investigation of heart failure with congestion and heart failure of anginal (ischæmic) type we are now in a position to review some of the recognizable factors upon which the prognosis is determined in defined varieties of cardiac disorder.

CARDIAC ARRHYTHMIA.

Knowledge of the significance of cardiac arrhythmia was placed on a firm basis by Mackenzie, who was able, by means of the polygraph, to recognize two groups, which he designated: (i) the simple or harmless type, in which sinus arrhythmia and extrasystole were defined; (ii) the "dangerous type" of irregularity, which we now describe as auricular fibrillation. In this group also are to be included the rarer types of irregularity recognized by the electrocardiograph, such as heart block and auricular flutter.

Simple Arrhythmia.

Subsequent investigation has borne out the judgement of this great clinician in assessing the importance of these signs. In discussing the significance to be attached to the presence of extrasystoles, the present view is summed up by Lewis thus:

> When extrasystoles are found, the heart amongst other organs should be scrutinized closely. If no further signs are detected the extrasystole can be neglected; if other signs are found the prognosis will be based on these.

In general when one is confronted with extrasystoles, careful investigation should be made to eliminate focal sepsis, tobacco poisoning, digestive disorders and sources of reflex irritation. Their presence as an isolated phenomenon is unimportant from the viewpoint of prognosis.

Dangerous Arrhythmia.

Auricular Fibrillation.

The mechanism of auricular fibrillation can be determined with certainty by the electrocardiograph, although in most instances this disorder of rhythm is recognizable clinically. In difficult cases associated with a high degree of heart block the sphygmomanometric test is often of value, determining as it does the varying levels of blood pressure at which the irregular beats come through to the radial pulse.

The presence of the "dangerous type of irregularity" is usually significant of organic disease if persistent, indicating structural damage to the conducting mechanism. That this is not necessarily so, however, is indicated in cases of paroxysmal fibrillation and flutter, in cases of fibrillation which may be made to revert to normal rhythm by the use of quinidine, and by the disappearance of the mechanism of auricular fibrillation after the removal of a toxic goitre.

Heart Block.

Heart block is usually of grave prognostic significance (excepting when it arises as a drug effect), indicating as it does structural change causing interference with the conducting mechanism.

Pulsus Alternans.

Pulsus alternans is a type of irregularity frequently recognizable clinically or by the sphygmomanometer, or sometimes detected electrocardiographically, although the electrocardiograph is not infrequently dumb in these cases. When persistent and associated with slow rates of the heart, it is usually of serious prognostic significance, and is thought to indicate that on alternate beats portion of the fibres of the ventricle fail to contract. Occurring in association with high rates of the heart, as in auricular flutter or in paroxysmal tachycardia, it is frequently a temporary disorder and of less serious significance.

Treatment and Prognosis in Arrhythmia.

Although it is established that patients exhibiting the irregular action of auricular fibrillation are *par excellence* the most satisfactory subjects for digitalis therapy, the prognosis depends on the ability to maintain adequate circulation at a reasonable ventricular rate—80 to 90 per minute. Each patient offers special problems, and the duration of life is variable, depending largely on regulation of effort and the response to treatment.

> A patient recently observed in hospital maintained reasonable activity for twelve years after the original onset of failure with congestion at the age of fifty-four years. Digitalis was administered practically continuously during this period.

Of the rarer irregularities referred to it may be said that their significance lies in the fact that they emphasize the importance of the search for other evidences of disease which may determine the prognosis.

HYPERTENSIVE CARDIO-VASCULAR DISEASE.

The recognition of hypertension or high blood pressure is usually an incident in the course of routine investigation or examination of persons for life insurance. . Apart from epistaxis or other bleedings, there are really no symptoms peculiar to hypertension, although nervous symptoms, irritability of temper, emotional upset, failure of memory and concentration, headache following exercise, throbbing in the head *et cetera* are frequently complained of by the patient.

In the assessment of the prognostic significance of high blood pressure it is important to observe in particular whether there is associated evidence to suggest incipient circulatory failure, cardiac hypertrophy or arterial degeneration.

It is important to recognize that emotional stress and tension frequently give rise to temporary high blood pressure records, which are of much less grave prognostic significance than continued hypertension, particularly with an associated elevation of diastolic pressure. It has been stated that Australian figures from the experience of insurance companies indicate that systolic readings in this community are frequently 10 to 12 millimetres of mercury higher than the accepted standards in England and the United States of America.

In cases in which the diagnosis of hypertension is established, particularly when associated with cardiac hypertrophy and evidence of early arterial changes, it must be recognized that the expectation of life is distinctly lessened, the disease tending to progress, usually little controlled by treatment beyond restriction of major activities and general regimen of health, and frequently terminating by vascular accident or leading to coronary disease, and in many cases merging insidiously into arteriosclerosis. The prognosis in cases of essential hypertension is, however, apart from unforeseen vascular accidents, largely determined by evidence of cardiac failure.

It is probable that medicinal treatment has little effect in such cases beyond benefit derived from the inculcation of a moderate régime, imposing adequate rest, gentle exercise and avoidance of all excesses. The judicious employment of "Luminal" or similar mild sedative appears to be the limit of effective medicinal treatment. The multitude of drugs introduced for the treatment of hypertension from year to year form a striking testimony to their therapeutic uselessness.

Recently various surgical procedures have been advocated in the treatment of essential hypertension; these measures are as yet incompletely standardized.

In cases in which there is evidence to suggest that arterial damage has occurred, the electrocardiograph is frequently of value in indicating damage to coronary vessels and consequent myocardial disease.

CARDIO-VASCULAR SYPHILIS.

Syphilis as an ætiological factor in the production of cardiac disease has only of late years become recognized in its full and baneful significance, although its predominance in the causation of aneurysm had long been established. Analysis of the incidence of syphilis as the essential factor in cardio-vascular lesions has recently revealed the astounding fact that in all adult cases of cardiac failure (unselected) at least 5% are specific in origin, the figures rising as high as 15% in some series of observations reported by Carey Coombes. The position may be summarized by saying that when we are confronted by an organic disease of the heart in an adult there is probability of a specific origin in 5% to 15% of all cases. If a selected lesion (aortic aneurysm) be considered, the incidence of syphilis is stated to be 75% to 83%.

The percentage of syphilis in the common forms of cardiac breakdown is given in Table I.

TABLE I.
The Incidence of Syphilis in Cardiac Breakdown.

Disease.	Percentage Due to Syphilis.
Aneurysm	75
Aortic disease	30
Cardiac pain (transient)	15
Cardiac infarction	1 to 3
Ventricular failure	20
Heart block	10
Auricular failure	2

Syphilitic aortitis is the characteristic cardiac lesion of specific carditis. The inflammatory process tends to concentrate upon the aorta at its origin, and early in its course comes to involve: (i) the mouths of the coronary arteries, (ii) the semilunar valves.

Symptoms and Signs.

From the viewpoint of prognosis it is important that the possibility of syphilis in cardiac disease should constantly be borne in mind. The symptoms and signs are classically as follows:

The patient is usually a man in the middle period of life, aged forty-five to fifty years, complaining of breathlessness on exertion, and a deep-seated substernal pain activated by exertion, but not infrequently appearing while at rest. Hypertension is generally present, and usually there is a greatly increased pulse pressure.

Physical examination at this stage reveals cardiac hypertrophy and a forcible apical impulse. Evidence of aortic valvular leak may be found. The blood pressure is usually raised in company with the increase in pulse pressure, for example, 180 millimetres of mercury (systolic) and 70 millimetres of mercury (diastolic). Radiographical examination usually reveals an enlarged heart, frequently with evidence of aortic dilatation.

Syphilitic infection has usually occurred 15 to 20 years previously. Collateral evidence of this infection may or may not be discernable. There is usually a reaction to the Wassermann test.

Prognosis.

The general experience of observers is that syphilitic aortitis is necessarily a progressive and fatal disease, the average duration of life after the condition has been recognized clinically being usually five or six years.

In some instances improvement has been recorded after vigorous antispecific treatment. Thus is

stressed the importance of the earliest possible recognition of this factor in cardiac breakdown.

SUBACUTE BACTERIAL ENDOCARDITIS.

The recognition of this malady is of prognostic importance because the prognosis is perhaps more clearly defined than in any of the diseases of the heart which have been considered. The disease runs a varied course, but almost invariably proceeds to a fatal termination irrespective of treatment. Early clinical recognition is therefore of importance. It is recognized that the malady usually commences insidiously as a low-grade septicæmic infection in adult life, lassitude, weakness, malaise and occasional feelings of chilliness being the initial symptoms, leading to an acute febrile illness with toxic signs and frequently embolic manifestations. Frequently there is evidence of preexistent valvular disease. The petechial spots commonly observed are due to increased permeability of capillary vessels resulting from toxæmia. Congestive cardiac failure or embolic complication usually determines the method of death.

RHEUMATIC CARDITIS.

The modern conception of acute rheumatism is that of a systemic invasion by an acute infective agent, causing damage to the small arterioles of the body, setting up a process of inflammation acute or chronic, and usually manifested by evidence of disease in and around the joints, in the heart and in subcutaneous tissues and tendons.

The changes produced in the heart are conveniently referred to as rheumatic carditis. The disease is particularly one of childhood, exhibiting a strong tendency to relapse or to run a chronic course, with exacerbations, resulting in permanent damage to the structure of the heart. In the case of a child smitten with rheumatic fever the prognosis is dependent upon: (i) the initial damage done to the heart and the consequent changes in its structure, (ii) the tendency to recurrent infection with subsequent damage. Ultimate prognosis is therefore difficult and uncertain in initial attacks, and the outlook becomes progressively worse after each subsequent recrudescence of infection. In cases in which recurrence is frequent and attacks are severe, the patient is likely to succumb before adult life is reached.

In the case of an adult who has survived rheumatic infection of childhood the outlook is dependent upon the amount of residual cardiac damage. In such instances it should be remembered that the development of valvular disease, such as mitral stenosis, is insidious, progressive fibrous tissue change continuing for a long period after the phase of active infection has been passed. It is therefore in cases of established valvular disease of rheumatic origin in adults that prognosis becomes of the greatest import, so that a measure of useful activity may be enjoyed by such individuals.

In assessing the outlook for patients who are the subjects of established rheumatic cardiac disease it is customary to recognize certain clinical types.

1. In those cases in which damage to valves is the prominent recognizable sign, valvular disease, mitral stenosis or aortic regurgitation or stenosis is recognizable without gross cardiac enlargement or evident restriction of cardiac response to effort. In such cases the outlook is relatively hopeful, and a useful and active life is frequently enjoyed. The possibility of a superimposed bacterial endocarditis at a later period is a possible contingency.

2. In the second group irregularity of heart action due to auricular fibrillation is present; breathlessness is complained of on severe exertion. Evidence of a minor grade of mitral stenosis is usually found without gross cardiac enlargement. Such patients are usually well controlled by digitalis and may continue for several years at a moderate grade of activity.

3. The third group comprises the greater number of patients who suffer severely from breathlessness. Exercise tolerance is poor. Cardiac enlargement is usually considerable, and auricular fibrillation is usually present in association with mitral stenosis. The prognosis is less favourable than in the preceding groups, and a life of restricted activity is necessarily enjoined, cardiac failure being likely to supervene. It is stated that one-third of such patients succumb in ten years.

4. In the fourth group there is evidence of more grievous cardiac damage, and gross enlargement is present, often associated with angina of effort. In other cases congestive failure is manifest, and in these the immediate prognosis depends upon the response to digitalis.

It is apparent that hard and fast rules for prognosis in the rheumatic group of cases are difficult to define and that a summation of the factors peculiar to the individual case must be taken into consideration in estimating the outlook.

ARTERIOSCLEROSIS AND SENILE DEGENERATIVE DISEASE.

It has been aptly remarked that a man is as old as his arteries. In many individuals the process of wearing out is made manifest by investigation of the cardio-vascular system. It is obvious that, as the elasticity of blood vessels diminishes with advancing years, so does the efficiency of the circulation become impaired. Clinically one recognizes arterial disease by observing the prominence of vessels in their course and noting their tortuosity. Under normal conditions the arteries are not visible in the healthy young adult. The earliest sign of thickening and loss of elasticity can usually be found by observing the brachial artery in its course. Thickening of the radial artery can be appreciated by rolling the vessel (emptied of its blood by compression) under the finger-tips. The early stages of thickening of the vessel wall can be appreciated in this manner. Ophthalmoscopic examination of the retinal vessels is also of practical value to the clinician. The fluoroscopic picture of the arteriosclerotic heart is also of extreme importance.

In assessing the results of arterial thickening it is important to remember that the material results of arterial degeneration are interferences with nutrition of tissue.

The nutrition of cardiac muscle, and hence the determination of myocardial degeneration, is therefore dependent upon the state of the coronary vessels. In the clinical recognition of these changes, restriction of cardiac response to effort, shortness

of breath and the production of cardiac pain by relative ischæmia of the heart muscle have already been described in the review of the evidences of failure of function of the cardiac mechanism. In cases of degenerative disease considerable evidence of structural change is frequently derived from the study of the heart by electrocardiography. Coronary sclerosis, branch bundle block, arborization block, and other evidence of alteration of myocardial structure, can frequently be demonstrated. It is well to remember that patients with branch bundle block are particularly liable to sudden and unexpected death.

Coronary stenosis leading to acute coronary obstruction is now well recognized clinically and by the electrocardiograph. Apart from a sudden accident of this type, carrying as it does an immediate threat to life, the prognosis in degenerative vascular disease as a whole is to be assessed on a careful survey of all the evidence, clinical, electrocardiographic, radiological, and has frequently to be correlated with the evidence of nutritional failure in the other organs, arteriosclerosis of the kidneys, and general vascular degeneration.

Principles in Prognosis.

From this review of some of the types of cardiac disorder leading to failure certain principles emerge for our guidance in prognosis. In deciding that a patient's heart is essentially sound the most valuable indication is the evidence of the power to engage fully in his work without symptoms. No patient with normal capacity for full bodily exercise is likely to have grave heart disease.

Anginal pain arises as a result of inadequacy of blood supply to the heart itself.

Good exercise tolerance is a reliable guide in excluding serious cardiac disease, provided cardiac enlargement, evidence of mitral stenosis, aortic regurgitation, auricular fibrillation, hypertension and congenital anomalies are excluded by physical examination aided by fluoroscopy and electrocardiography. In the systematic examination of cardiac patients investigation should proceed along well-directed lines: (i) exercise tolerance, (ii) congestion, (iii) enlargement, (iv) rhythm, (v) condition of the valves, (vi) infection, (vii) vessels, (viii) ætiology.

In the assessment of prognosis where the patient is suffering from chronic heart disease the guiding considerations should be the following: (i) the functional efficiency of the circulation (breathlessness at rest or produced by exercise, the presence or absence of anginal pain on effort); (ii) the size of the heart; (iii) the presence or absence of syphilitic aortitis; (iv) the presence of gross disturbance of rhythm (auricular fibrillation, heart block *et cetera*); (v) the presence of hypertension; (vi) evidence of structural disease of valves or myocardium. Acting in accordance with these principles it is in most instances possible to place the patient in one of the following categories (taken from Lewis's "Diseases of the Heart").

A. Good Expectation of Life.
- Valvular disease in young adults, without auricular fibrillation, and with good exercise tolerance.

B. Several Years' Expectation (about ten years).
- Angina of effort (early years).
- Hypertension, with little enlargement, fair tolerance.
- Auricular fibrillation with or without valvular disease, but little enlargement and fair tolerance.
- Enlargement moderate, with or without valvular disease, with fair tolerance.

C. A Few Years' Expectation (three to six years).
- Angina of effort on walking a short distance, with or without previous coronary thrombosis.
- Syphilitic aortitis.
- High tension, with moderate enlargement or angina.
- Enlargement, great or moderate, with poor tolerance.
- Auricular fibrillation and moderate enlargement.
- Failure with congestion (relieved by treatment).

D. Living Precariously up to One or Occasionally Two Years.
- Enlargement great—congested.
- High tension and cardiac asthma.
- Congestion unrelieved or in syphilitic aortitis, in high tension, following angina or after coronary thrombosis.
- Angina unrelieved by rest.

E. In Imminent Danger.

Œdema of lungs. Coronary thrombosis (recent). Adams-Stokes syndrome (epileptic state).	Often recovering.
Leaking aneurysm. Anginal state. Gross failure with congestion (unrelieved).	Rarely or never recovering.

Epilogue.

In cardiac disease, as indeed in all human ailments, the practice of our art is far removed from the precision of an exact science. Diagnosis depends on the observation of symptoms and physical signs, prognosis upon the evaluation of their significance, based on experience and modified by the individual characteristics of the patient.

In no branch of medicine is the Hippocratic axiom more apposite:

Vita brevis. Ars longa. Occasio celeris. Experimentum periculosum. Judicium difficile.

The experience of a lifetime is all too limited, and judgement amazingly difficult.

SOME OBSERVATIONS UPON THE EXAMINATION OF MEN EXPOSED TO A LEAD HAZARD AT MOUNT ISA, INCLUDING AN ORIGINAL METHOD FOR THE EXAMINATION OF BLOOD FOR POLYCHROMASIA.

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Source of Material.

The material which forms the basis of these observations was collected by the writer from 115 men employed by Mount Isa Mines, Limited, in one or other of the departments, who, for some

reason or other, consulted the chairman of the medical board with a view to ascertaining whether or not they were suffering from plumbism. In most instances the men were complaining of symptoms thought by themselves to be attributable to lead poisoning. Some patients came merely as a matter of routine. Only one of these men was considered to be suffering from lead poisoning as distinct from mere lead absorption (that is, to be incapacitated by plumbism), and in this case the lead poisoning was complicated by duodenal ulcer. This case is interesting from the point of view of the contention of Peipers that "lead poisoning may produce the conditions that lead to the development of gastric ulcer". For the most part the various symptoms, mostly vague, of which complaint was made by these men were thought not to be caused by lead.

In addition to the examinations carried out by the chairman, which form the basis of the following observations, all the employees of Mount Isa Mines, Limited, are examined regularly by the company's medical officer.

Most of the men in this group were in their twenties or early thirties. The average age was thirty years; the oldest man was aged fifty-two and the youngest twenty-one years. There were several of the latter age. The men under consideration are thus seen to be young men. The majority of them were single.

The average time that these men had been employed in a lead hazard was two and a half years, varying in individual cases, however, from four months to eight years. The largest number of men were employed in the mine, almost as many in the smelters, and quite a minority in the mill or in the mechanical sections.

The magnitude of the lead hazard to which these men were subjected was probably not of a very high order, as evidence the fact that of 1,231 men employed in all departments, only four were considered by the medical board to be incapacitated by plumbism during the first half of 1937, and only two in the second half of the same year.

SYMPTOMS.

The symptoms of which complaint was made by the men were mostly of the vaguest nature and for the most part do not merit consideration, being indicative of no more than general malaise, the cause of which might have been anything, particularly in a climate such as that of Mount Isa, where the temperature is frequently excessive for long periods, so that most people feel below par. Perhaps two symptoms warrant further notice.

Constipation.

Constipation was a fairly constant symptom, 22 of the men complaining of slight constipation, and 15 more of greater degrees of the same symptom. It would thus seem that constipation was a frequent accompaniment of lead absorption, and, as far as lead poisoning is concerned, one would hesitate to make the diagnosis in the absence of constipation.

So-Called Arthralgia.

Twenty-two of the men complained of pain more or less definitely located to one or more joints. It is felt that it is difficult to assess the value of this symptom, or to distinguish it from the aches and pains that accompany the small traumata more or less the inevitable accompaniment of an active labourer's life. It is, furthermore, not always easy to be sure that a man's pain is actually in his joint, or whether it is not rather periarticular in distribution. It is thought that the pain called "arthralgia" which accompanies true lead poisoning is periarticular pain.

Other Symptoms.

The other symptoms about which complaint was most frequently made were headache, loss of appetite, loss of energy, lassitude, nausea and, not infrequently, giddiness. No unequivocal case of colic was encountered.

SIGNS.

Burton's Line.

Burton's line was observed in only five cases, in two of which it was very slight. For the most part the oral hygiene of the men was good, and this possibly explains the infrequency of this sign. In looking for the sign only naked-eye examination was employed. It is possible that if a lens had been used the sign might have been observed more frequently. On the other hand there is no lack of good light at Mount Isa.

Blood Pressure.

The average systolic blood pressure in the group was 125 millimetres of mercury, while the average diastolic pressure was 80 millimetres of mercury. The highest pressure recorded was 155 millimetres of mercury (systolic) and 95 (diastolic) in a man, aged fifty-one years, who gave a history of chronic alcoholism, and was thought to be suffering from alcoholic peripheral neuritis. His arteries were thickened to the touch. There were two other men with a systolic blood pressure of 150 millimetres of mercury, and one of these was of an extremely nervous temperament, which may explain the reading. There were also two men with a systolic blood pressure of 140 millimetres of mercury. One of these two was suffering from diphtheria, and the other had thickened arteries and rheumatoid arthritis. These figures would tend to confirm Teleky's contention that "it has never yet been scientifically demonstrated that increased blood pressure accompanies signs of fresh lead absorption".

It should be noted, however, that this group consists mostly of young men, whereas, as pointed out by Teleky, it is desirable that in a consideration of this nature the men should be over forty-five years of age, "since at this age vascular changes stand out more plainly and the blood vessels are more sensitive to injury".

STRENGTH OF HAND GRIP.

Using a dynamometer of the Collins type and taking the readings on the *échelle de traction*, the calibration of which is doubtful, I found the average reading for the right hand to be 85, and that for the left 80. The lowest readings recorded were right 25, left 20; and the highest right 125, left 115. Contrary to the opinion of Sir Thomas Legge that the strength of the hand grip is the most generally useful test in the search for lead poisoning, as far as estimating the strength of the hand grip by using this kind of dynamometer is concerned, it is felt that the test is thoroughly unreliable. There is no guarantee that the subject is using the maximum effort of which he is capable. For instance, the lowest reading just quoted, namely, 25 and 20, was that of a man applying to the board for compensation. A few months before, when there was no question of compensation, the reading obtained was 95 (both hands). Apart from this subjective aspect of the readings, another quite important factor is the hardness of the hand. It would seem that in many cases the maximum reading is determined by the pain produced by the instrument, rather than by muscle strength. And, finally, there is a definite knack which may be acquired whereby by a sudden squeeze a momentarily high reading may be obtained by those who have learnt the trick.

BLOOD FINDINGS.

Hæmoglobin Estimations.

In common with those of other observers, the findings suggest that the Tallqvist scale is practically useless for readings above 60%, for it fails to correspond even approximately with more accurate methods.

The instrument used in making these estimations was a Helligë "Neoplan Hæmometer". The average reading obtained was 94% on the "Neoplan" scale, the lowest being 72% and the highest 112%.

It is felt that an estimation of the hæmoglobin content of the blood gives no indication of lead absorption, but that in most cases of developed poisoning with incapacity the value is below normal.

Ratio of Large to Small Lymphocytes.

In the writer's hands the test for the estimation of the ratio of large to small lymphocytes has been found disappointing and has been abandoned as of little or no value. The chief criticism offered concerning this evaluation is that if a division of the lymphocytes is made merely upon a consideration of absolute size (for example, ten micromillimetres), then too many uncontrollable factors are involved for the estimation to be of general use, however useful, which I doubt, it may be in individual hands. The spreading of the blood film makes a vast difference in the apparent size of all the blood cells, and the lymphocytes are no exception. Even on the one film the ratio of large to small lymphocytes I have found to vary as much as from 9·0 in a thin portion of the smear to 1·1 in a thick portion of the same smear. However skilful one may be at spreading blood films, there is no guarantee that the amount of spreading will always be the same.

Possibly the method used by Ferguson, in which the size of the lymphocytes is compared with that of the erythrocytes, may give more reliable results; and possibly a method in which hæmatological morphology rather than mere size was used to distinguish between large and small lymphocytes would be an improvement.

In any case, if the staining methods are good, it is not easy to accept the statement that it is difficult to distinguish a large lymphocyte from a monocyte.

Stippling of the Red Cells.

As has been previously observed, when a light-field method was used, there was no significant difference in the counts obtained whether Giemsa or Sellers's stain was used. For the most part use was made of the latter.

The average count obtained was 110 (small and large granules) stippled cells per million erythrocytes. The maximum count was 2,000. It will be seen that, unlike the findings of some other observers, including Lane, my counts have been on the low side, in many instances the count being zero.

The counts obtained were distinctly higher during the cool months of the year. In the months May to September the average count was 210 as compared with 50 for the remainder of the year. Since conditions are very hot during this latter period, the temperature of the tap water used for washing being, for instance, frequently in the region of 37·8° C. (100° F.), and although the Sellers's stain used was made up frequently and kept in the refrigerator when not in use, it is thought that, in the absence of any other obvious reason for the discrepancy, some technical error, probably having to do with the temperature, is involved. This aspect might well be investigated.

Stippled cell counts, by means of the dark-field method introduced by Nelson, were also used, and the average count obtained was 1,300 (small and large granules) stippled cells per million red cells. The maximum was 10,000 and the minimum nil. As with the light-field method, the count was higher during the colder months of the year, and probably for the same undetermined reason. The average count for the months May to September was 2,000 and that for the months October to April was 760.

Diffuse Polychromasia.

As regards the place of polychromasia in the diagnosis of lead poisoning, probably the dictum of Lane can be accepted.

The stippled cell is the young cell; and the close connexion between reticulocytosis, polychromasia and punctate basophilia is now generally accepted. Reticulocytes are susceptible to injury, and as a result of damage, their reticular substance may become so altered that it becomes visible by the ordinary staining methods; these cells now display polychromasia or punctate basophilia. The typical reaction resulting from the action of lead

is that of punctate basophilia, but frequently, following lead exposure and before the appearance of stippled cells in the blood, a high polychrome count is the only change found.

Method of Examining for Polychromasia by Dark Field.

In the course of the examination of blood films by the dark-field method of Nelson, using Sellers's staining method, I observed that polychromatic cells also could be distinguished by the same dark-ground method. The method finally adopted was as follows.

Ordinary thin blood films are made on clean, thin (1·0 millimetre or less) microscopic slides in the ordinary way. After drying, the films are fixed by running pure methyl alcohol on the slide, film up, and allowing it to remain for two minutes. The film is allowed to dry in the air, and then Sellers's stain is run on and allowed to remain on the film for forty-five seconds. The stain is then washed off with running tap water, and the washing continued for one minute. The film should then be very pale green in colour. It is allowed to dry in the air and is then examined by the dark-field method.

In the microscopic examination the source of light used was a Zeiss "Sphärolux" projector lamp used without the blue glass filter. The microscope was a Zeiss binocular, used with a Zeiss "Cardioid" dark-ground condenser. The objective was a × 60 oil-immersion apochromatic lens used at full aperture, namely, a numerical aperture of 1·0, which was found to give the best results. The oculars usually employed were "Mobimi" (× 10, sometimes × 7). Distilled water was used between the condenser and the under surface of the slide, and Martindale cedar-wood oil for the immersion of the objective.

By this method of examination the ordinary orthochromatic cells show up as yellow rings, while the polychromatic cells are pink to reddish-pink rings. The number of such polychromatic cells in fifty fields was counted and the number of erythrocytes in each field was estimated by the method described by Badham.

That the pink cells seen by this method actually correspond to the polychromatic cells as seen in the light field was frequently verified by changing over from dark field to light field, when individual cells which had this pink colour in the dark field were shown to be polychromatic in the light field and *vice versa*.

Just as for light-field examination, the staining has to be carefully controlled. If the film is overstained all the cells appear pink, just as they all appear blue under the same conditions in the light field. It was found necessary to make up the Sellers's stain at least every three weeks, and it was kept in the cold when not in use. If the stain is allowed to become too polychromatic it is found that the film when washed will not become green, but remains blue, and then all the cells are pink or even red in the dark field.

Although the Romanowsky stains, Giemsa or Leishman, especially Giemsa, are very suitable for the light-field examination for polychromasia, they did not give a sufficiently clear differentiation in dark-ground work.

The counts obtained with Giemsa, with Sellers's stain examined under the light field, and with Sellers's stain examined by the dark field correspond quite well, as is seen in Table I, which shows the comparative counts.

TABLE I.

Number of Polychromatic Cells per Million Erythrocytes.

Light Field.		Dark Field.
Giemsa Stain.	Sellers's Stain.	Sellers's Stain.
—	250	0
—	0	0
—	170	0
—	200	0
—	0	0
1,300	2,200	1,200
—	0	0
—	450	0
—	0	0
—	230	0
4,800	3,800	3,100
1,000	0	670
730	400	250
—	0	0
390	830	1,000
150	0	200
170	0	0

Taking into account errors due to sampling, and remembering that a count up to 250 per million usually means that one polychromatic cell has been seen, the correspondence will be seen to be reasonably close, and it is safe to assume that the dark-ground polychromatic cell count is at least of the same order as the light-field count.

With practice the polychromatic cells, like the stippled cells, are picked out more readily in the dark field, and a dark field is most certainly very much easier on the eyes of anyone who must do large numbers of counts during a day.

The average count of polychromatic cells was 460 per million erythrocytes, the highest count being 4,800 and the lowest nil.

SUMMARY.

1. Some observations concerning common signs and symptoms found in 115 men exposed to a lead hazard are recounted.

2. Blood findings are also considered.

3. Criticisms of the estimation of the ratio of large to small lymphocytes as a practical laboratory method, and of the common method of estimating the strength of the hand grip are advanced.

4. A dark-field method of examining red cells for polychromasia is described.

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ENDEMIC TYPHUS IN NORTH QUEENSLAND.

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Introduction.

FEVERS of the typhus group are endemic in north Queensland. On the one hand are those characterized by the development of specific agglutinins for *Bacillus proteus OX*19, which correspond to the urban flea-borne typhus of other parts. On the other hand are those characterized by the development of specific agglutinins for *Bacillus proteus OXK*. In this group some exhibit a cutaneous inoculation lesion resembling exactly the eschar of mite-borne Japanese river fever. More, however, resemble the rural or scrub typhus of the Malay States in that a cutaneous inoculation lesion is not evident. Previous reports which established Mossman, coastal and other previously unclassified fevers of north Queensland as endemic typhus have been communicated by Langan and Mathew (THE MEDICAL JOURNAL OF AUSTRALIA, August 3, 1935, page 145) and Unwin (THE MEDICAL JOURNAL OF AUSTRALIA, September 7, 1935, page 303).

The following observations are based upon data procured in the investigation of fevers occurring in the coastal strip of north Queensland between Tully and Daintree, and on the contiguous area of the Atherton Tableland. The period covered is the two years from May, 1935, to April 1937. During that period investigations of some sort were made in 309 cases of fever. Conclusive laboratory tests were obtained in confirmation of endemic typhus in 77 cases and of leptospirosis in 21 cases. These figures do not in any way indicate the percentages nor necessarily the relative frequency of occurrence of the two diseases in the total number of cases. In many cases which were undoubtedly endemic typhus the only blood samples available for serological tests were taken before agglutinins had developed. It is equally certain that many mild cases that could not be fully investigated were forms of leptospirosis. In the detailed analysis only cases of endemic typhus which could be adequately confirmed are included. The data procurable in regard to these varied from full records to meagre statements, and the analyses have been consequently restricted.

Most of the Cairns patients, and some of the outside patients were seen by me, and information was kindly given to me by the respective medical attendants.

Physical and Economic Features of the Area.

The area of country is roughly a semicircle, of 70 miles' radius, lying west of and radiating from Cairns. Cairns, a seaport of 13,000 inhabitants, is the largest centre of population in the area, and is situated 16° 55′ south of the equator, and 145° 46′ east of Greenwich. The anual rainfall in the area varies from about 80 inches to 150 inches or more. The rainy season is in the summer and generally lasts from December until April. During these months it is hot and humid on the coastal strip. In the winter little rain falls, the days are clear and warm, and the nights are fresh. The coastal strip is mainly fertile scrub land, much of which has been cleared and cultivated, the main crop being sugar-cane.

The main range rises sharply at distances of from one to twenty miles from the coast, the altitude at Atherton, in the centre of the Atherton Tableland, being 2,466 feet.

The climate on the tableland is equable and bracing. Minerals, including gold, are found throughout the area, a great deal of valuable timber is cut and marketed, tropical fruits are grown, and on the tableland large areas are devoted to maize-growing and dairying.

The population of the area is about 65,000. Most of the people are Australian born, of British stock. There are many southern Europeans engaged in the sugar industry, a sprinkling of Asiatics, and several hundred Australian aborigines, mainly living in mission reserves.

None of the patients who reacted to *Bacillus proteus XK* was an Australian aboriginal, one patient from the Yarrabah Mission area being the wife of an official in charge of an out-station.

TABLE I.
The Numbers of Cases According to Localities.

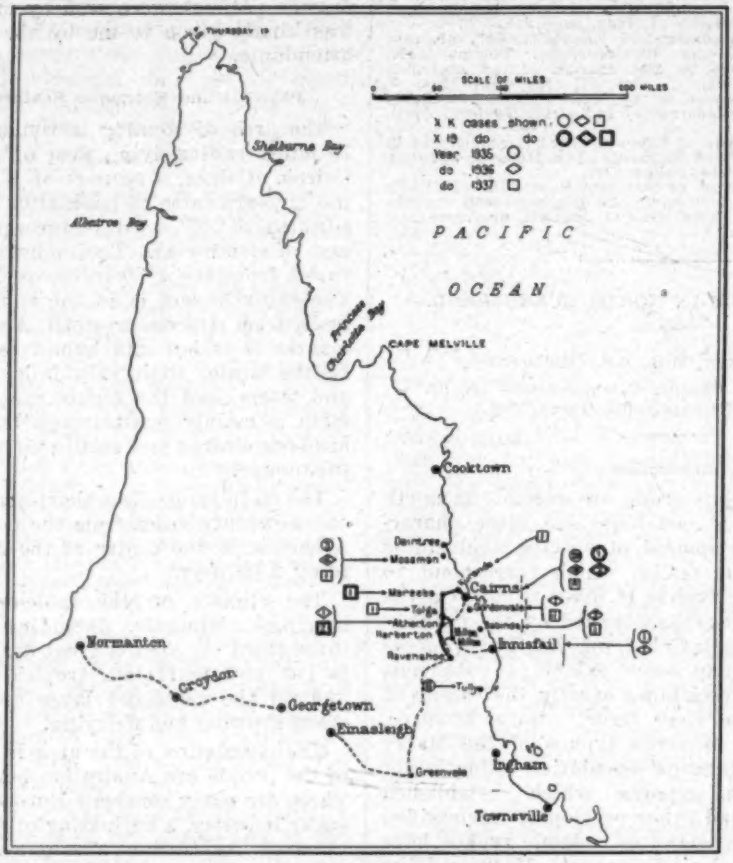
Locality	1935.		1936.		1937.		Total.	
	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.
Cairns	1	20 (2)	2	18 (1)	—	6	3	44
Gordonvale ..	—	—	—	1	—	1	—	2
Babinda ..	—	—	—	2	—	1	—	3
Innisfail ..	—	1	—	1	—	—	—	2
Tully	—	8 (1)	—	—	—	—	—	8
Mossman ..	—	3	—	4	—	1	—	8
Kuranda ..	—	—	—	—	—	1	—	1
Mareeba ..	—	—	—	—	1	—	1	—
Tolga	—	—	—	—	—	1	—	1
Atherton ..	—	—	—	1	2	—	2	1
Millaa Millaa ..	—	1	—	—	—	—	—	1
Total ..	1	33	2	27	3	11	6	71

(NOTE.—Figures in parentheses indicate fatal cases.)

Distribution of Cases according to Locality and Period of Year.

In Map I the cases are shown according to localities, and in Map II the distribution of cases in the Cairns district is shown. The former is summarized in Table I and the latter in Table II. In Table III the distribution of cases by months is shown.

Though more cases, and the fatal cases, occurred towards the middle of the year, no significance can be attached to this fact, as the numbers are small and cases occurred in each month of the year. More men are employed on farms, on road-making, scrub-clearing and timber-getting during the cooler and drier months, and the occurrence of a greater number of cases of the *XK* type is to be expected. In the Cairns area there are at least four clearly defined localities which may be considered as foci where infection of the *XK* type occurs, namely, West Cairns and Edge Hill, Edmonton and Hambledon, Intake and Redlynch, Double Island and Yorkies Knob.



MAP I.

The Intake-Redlynch Focus.

From the township of Redlynch a road stretches inland for seven or eight miles along a valley into the foothills. It passes through fertile cane lands and then dense tropical scrub, roughly following the Freshwater Creek to the Intake, which is the source of the Cairns water supply. The nature of this country is shown in Figures I to IV. In 1927, when additions to the water scheme, involving scrub-clearing, were in progress, numerous cases of fever resembling the *XK* type of typhus occurred amongst the men employed. Since then cases have been occurring each year amongst the workers on the cane farms situated close to the scrub. From one farm there have been four cases of the *XK* type in 1935, five (one death) in 1936, and one in 1937. Generally the workers resided on the farm, though occasionally they lived in the township of Redlynch, going to and from their work daily. Of 28 rats trapped during September and October, 1936, on this farm and in the surrounding scrub, none had agglutinins

TABLE II.
The Distribution of Cases in the Cairns District.

Locality.	1935.		1936.		1937.		Total.	
	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.
City residents ..	1	8	2	3	—	3	3	14
Edge Hill and West Cairns	—	1	—	3	—	—	—	4
Intake and Redlynch ..	—	4	—	9 (1)	—	1	—	14
Edmonton and Hambledon	—	5 (1)	—	2	—	—	—	7
Double Island and Yorkie's Knob ..	—	2 (1)	—	1	—	1	—	4
Yarrabah ..	—	—	—	—	—	1	—	1
Total ..	1	20	2	18	—	6	3	44

(NOTE.—Figures in parentheses indicate fatal cases.)

TABLE III.
The Incidence by Months.

Month.	1935.		1936.		1937.		Total.	
	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.
January ..	—	—	—	—	1	2	1	2
February ..	—	—	—	2	2	3	2	5
March	—	—	—	1	—	1	—	2
April	—	—	—	1	—	5	—	6
May	1	1	2	1 (1)	—	—	3	2 (1)
June	—	16 (2)	—	—	—	—	—	16 (2)
July	—	9 (1)	—	3	—	—	—	12 (1)
August ..	—	—	—	2	—	—	—	2
September ..	—	3	—	8	—	—	—	11
October ..	—	2	—	2	—	—	—	4
November ..	—	3	—	3	—	—	—	6
December ..	—	1	—	2	—	—	—	3
Total	1	35	2	25	3	11	6	71

(NOTE.—Figures in parentheses indicate fatal cases.)

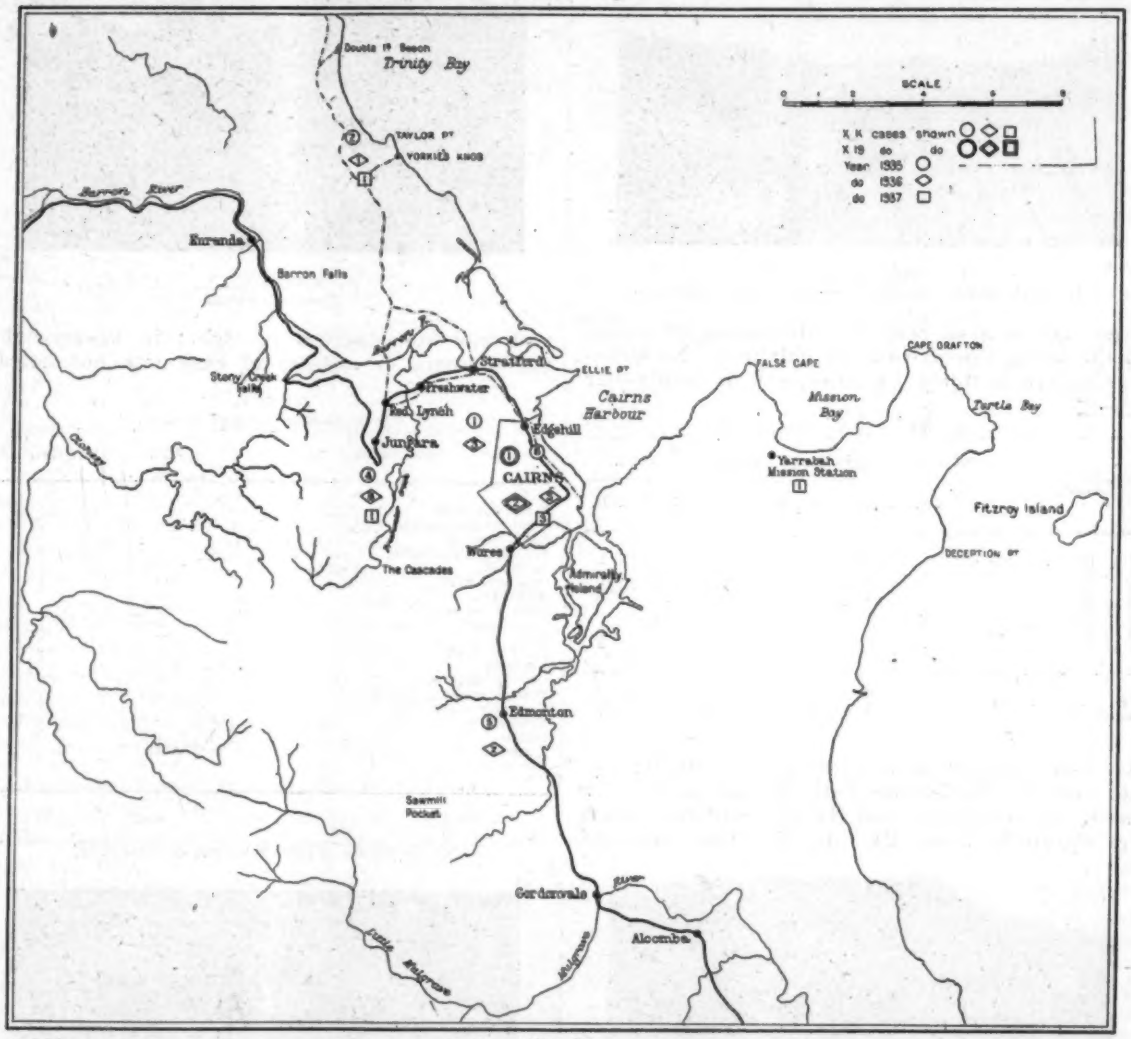
for *Bacillus proteus OXK* in a titre higher than 1 in 25.

Age, Sex and Occupation of the Patients.

In Table IV age and sex are shown. As might be expected, most of the patients were males, aged between twenty and forty years. The youngest was a girl, aged six years, suffering from the *XK* type.

Incubation Period.

In two instances, both fatal *XK* cases, an incubation period of thirteen days could be determined. In one of these there was a typical eschar on the back; in the other no cutaneous lesion of inoculation was found. In many other *XK* cases the incubation period appeared to be between nine and sixteen days.



MAP II.

A man, aged sixty-seven years, and a woman of the same age were the oldest; in both instances the infection was of the *XK* type.

In Table V the occupations are shown. Five of the patients with infection of the *X*19 type both lived and worked in towns. In most of the *XK* cases, such patients as cane-farm hands, timber-getters and scrub-cutters lived at or close to the places where they were working. In regard to those patients who neither lived nor worked outside the town, inquiry generally showed that visits to scrub or farm lands had been made a week or two before the sickness appeared.

Presence of Insect Bite or Eschar.

Bites of mites and ticks are of frequent occurrence amongst those moving about in the scrub lands. In seventeen *XK* cases a cutaneous inoculation lesion was present, resembling the characteristic lesion at the site of the mite bite of Japanese river fever. In some of these lesions, when first seen, a central, dark brown crust was observed; this later sloughed off, leaving a shallow, slowly healing ulcer, from five to ten millimetres in diameter. In others the ulcer only was observed. In one case at Mossman, in which there was a typical abdominal eschar with crust, a tick had

been removed from the skin at the site of the lesion some days previously. Ticks and mites were vaguely associated with the lesions in some other

FIGURE I.
Road to Intake passing through cane paddock.

cases; but in most cases no information in regard to the biting insect could be obtained. No lesion was noticed in thirty *XK* cases, and in twenty-four

TABLE IV.
Age and Sex of the Patients.

Age in Years.	1935.		1936.		1937.		Total.	
	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.	*X*19.	*XK*.
1 to 10 ..	—	—	—	1	—	—	—	1
11 to 20 ..	—	8	—	1	2	1	2	10
21 to 30 ..	—	10	1	8	—	7	1	25
31 to 40 ..	—	7	—	10	—	1	—	18
41 to 50 ..	1	5	1	3	—	—	2	8
51 to 60 ..	—	2	—	3	1	2	1	7
61 to 70 ..	—	1	—	1	—	—	—	2
Male	1	20	2	23	1	8	4	60
Female	—	4	—	4	2	3	2	11

XK cases the presence or absence of a lesion was not noticed. No lesions were present in the *X*19 cases. The situations and characters of the lesions are shown in Table VI. In all these cases the

FIGURE II.
Heavily timbered hillside at edge of cane paddock.

regional lymphatic glands were enlarged and painful. The cases occurred in various seasons of the year.

Presence and Character of Rash.

Rash was observed in all six of the *X*19 cases. Of the *XK* cases, rash was observed in thirty-five

FIGURE III.
Tropical scrub.

and was not observed in eight; in twenty-eight the presence or absence of rash was not noted.

TABLE V.
The Occupations of Infected Persons.

Occupations.	*X*19.	*XK*.
Cane-farm work	—	18 (1)
Road-making (scrub)	—	9 (2)
Scrub-clearing	—	5
Timber-getting (scrub)	—	3
Labouring	—	6
Cane-mill work	—	1
Hawking	—	1
Clerical work	—	1
Travelling	—	1
Shop work	—	2
Railway work	1	—
Butchery	1	1
Grocery store work	1	—
Peanut handling (bulk)	2	—
Reservoir supervision	—	1
Domestic service	—	5 (1)
School	—	5
Unemployed	1	4
Unspecified	—	8
Total	6	71

(NOTE.—Figures in parentheses indicate fatal cases.)

FIGURE IV.
Tropical scrub.

The day of appearance of the rash is shown in Table VII.

TABLE VI.
The Situation and Character of the Lesions.

Situation.	Eschar.	Ulcer.	Insect Bite.	(?) Tick Bite.	(?) Mite Bite.	Total.
Abdomen ..	4	—	1	2	—	7
Groin	1	—	—	—	—	1
Scrotum	—	1	—	—	—	1
Thigh	1	1	1	—	—	3
Chest	—	—	1	—	—	1
Shoulder ..	1	—	—	—	—	1
Axilla	—	—	—	—	1	1
Back	2	—	—	—	—	2
Total ..	9	2	3	2	1	17

TABLE VII.
The Time of Appearance of the Eruption.

Day.	X19.	XK.
Fourth	—	2
Fifth	2	23
Sixth	1	8
Seventh	1	1
Eighth	—	1
Not stated	2	—
Total	6	25

The character, distribution and duration of the rash varied within a wide range. The most common was of a bright red macular type, appearing on the trunk about the fifth day and extending to the limbs and sometimes the face. This lasted for five or six days and faded in another two or three days. In some the rash was morbilliform in character, and in others of a mixed type. Most of the cases in which no rash was observed were mild ones. A rash was generally present in the severe cases, though in one fatal *XK* case, in which the patient was of a dark-skinned type, no rash was observed. A typical eschar was present on the back, and death occurred on the thirteenth day.

Symptoms, Course and Duration of the Fevers.

In five of the *X*19 cases the onset was sudden; in the other case it was more gradual. Of the *XK* cases the onset was sudden in 33 and gradual in 10; in 24 the nature of the onset was not stated. In Table VIII the early symptoms are shown.

TABLE VIII.
The Early Symptoms.

Symptom.	X19.			XK.		
	Present.	Not Present.	Not Stated.	Present.	Not Present.	Not Stated.
Headache ..	6	—	—	59	—	12
Generalized pains	3	2	1	39	5	27
Photophobia ..	3	—	3	39	4	28
Conjunctival congestion	4	—	2	52	—	19
Sweats and shivers	5	—	1	26	9	36
Sore throat ..	—	—	6	7	10	54
Cough	—	—	3	10	11	50
Vomiting	—	—	2	14	18	39

As shown in Charts I, II and III, the fever is as a rule remittent in type and terminates by rapid lysis. In uncomplicated cases the pulse rate is relatively slow. Of the *X*19 cases, two were mild, three moderately severe, and one severe. Of the *XK* cases, 23 were mild, 22 moderately severe, 10 severe, and five of grave severity. There were four fatal cases, and in seven cases the degree of severity was not ascertained. Icterus was not evident in any. Some degree of mental affection, varying from apathy, dulness and drowsiness to a fully developed typhoid state, was present in most cases. Splenic enlargement was frequently noticed. Groups of lymphatic glands were enlarged and tender in 32 of the *XK* cases; no enlargement was observed in seven, and in 32 no record was made in regard to the lymphatic glands.

TABLE IX.
Duration of the Fever.

Duration in Days.	X19.	XK.
10	—	3 (2)
11	1	4
12	1	12 (1)
13	1	2
14	—	9
15	1	8 (1)
16	—	2
17	—	2
18	—	3
19	—	1
21	—	2
27	—	1
42	—	1
Not stated ..	2	20

(NOTE.—Figures in parentheses indicate fatal cases.)

The duration of the fever is shown in Table IX. Convalescence after the return of the temperature to normal was, as a rule, rapid. In the four fatal *XK* cases death resulted from increasing toxæmia and pneumonia. At *post mortem* examination in one of these cases hæmorrhagic consolidation of the left lung was found. The case and findings are fully described in *Health*, Volume XIV, Number 3, March, 1936. A notable exception to the rule of rapid convalescence was an *XK* case in which all symptoms had abated

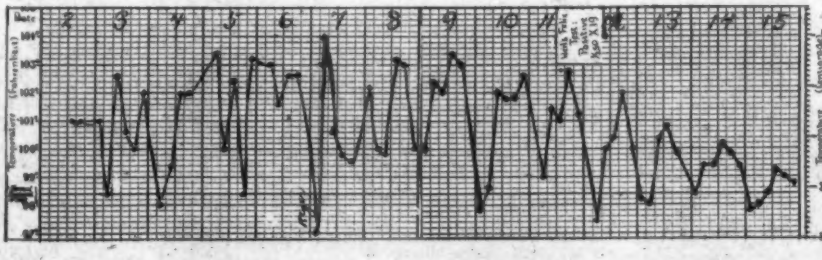


CHART I.
Temperature chart of a patient whose serum agglutinated *Bacillus proteus X*19.

after twenty-one days. There was, however, an evening rise of temperature to about 38·9° C. (102° F.) for the next twenty-one days. No focus indicating another infection could be found, and recovery was ultimately complete. The agglutination titres against *Bacillus proteus XK* in this case were nil on the fourth day, 1 in 500 on the thirteenth day, 1 in 1,000 on the twentieth day. There was no agglutination of *Bacillus proteus X*19.

Weil-Felix Tests and their Interpretation in Diagnosis.

In the earlier cases live suspensions were used for agglutination tests. From August, 1935, alcoholized suspensions were mainly used. The ideal of a series of agglutination tests with samples of blood taken at intervals could be attained in very few cases. A titre of 1 in 125 with the alcoholized suspensions was the minimum at which agglutination was regarded as a positive response to the Weil-Felix test. Exceptions to this rule have been two typical fatal *XK* cases and one typical but non-fatal *XK* case. In one of the fatal cases there were no agglutinins on the eighth day, and agglutination of *Bacillus proteus XK* at a titre of 1 in 50 was obtained on the twelfth day, when death occurred. In the other there were no agglutinins on the fourth or ninth day, and death occurred on the tenth day. In the non-fatal case the titres were nil on the second day, 1 in 25 on the eighth day, 1 in 50 on the eleventh day, 1 in 50 on the seventeenth day, all with *Bacillus proteus XK* only.

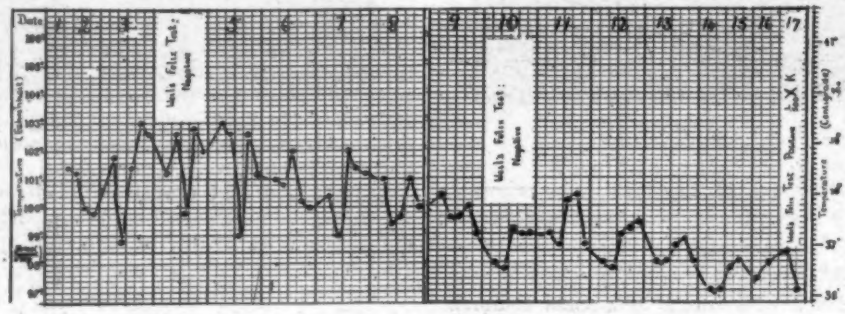


CHART II.

Temperature chart of a patient whose serum agglutinated *Bacillus proteus XK*. An eschar of the scrotum was present.

The highest titres that have been observed were 1 in 10,000 with *Bacillus proteus XK* and 1 in 1,000 with *Bacillus proteus X*19. Occasionally titres up to 1 in 25 with *Bacillus proteus X*19 were obtained in the *XK* cases; but no significant cross-agglutinations have been observed. The ninth day was the earliest day on which agglutination at a titre of 1 in 125 or over was observed. The characteristic rise of titre during the course of the disease and convalescence has been evident, though it has not been practicable to demonstrate the later fall in titre in many cases. In Table X the days and titres in regard to a number of cases are shown. Alcoholized suspensions were used.

Inoculation Tests.

Inoculation of Guinea-Pigs with Infective Material.

Guinea-pigs were inoculated with blood from three patients whose serum agglutinated *Bacillus proteus X*19. The earliest day of taking blood for the purpose was the seventh day. No scrotal swellings or other reactions were noted.

In 16 of the *XK* group of cases blood taken early in the disease (fourth to sixth day) was available, and guinea-pigs were inoculated. Mild febrile reactions were observed; but in none was there gross scrotal swelling.

Guinea-pigs were inoculated with brain material from a fatal *XK* case and scrotal swelling and fever developed. The findings were suggestive of infection with the virus of *XK* typhus; they are described in *Health*, March, 1936.

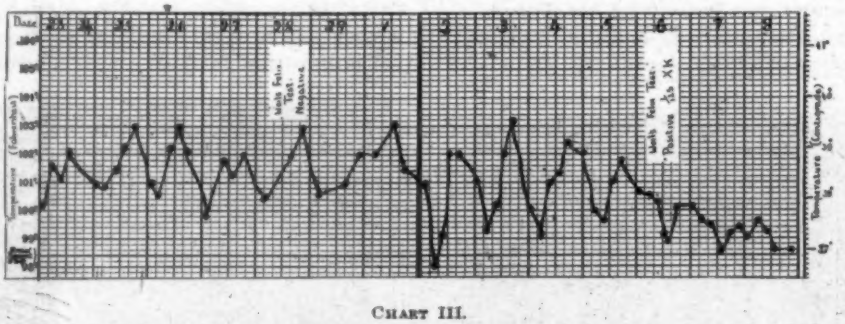


CHART III.

Temperature chart of a patient whose serum agglutinated *Bacillus proteus XK*. No eschar was observed.

Intraocular Inoculation of Rabbits with Blood from XK Patients.

During 1936 and 1937 attempts were made to infect rabbits with the *XK* virus by intraocular inoculation with blood from patients. The method as described by Lewthwaite and Savoor (*The British Journal of Experimental Pathology*, Volume XVII, February, 1936) has been followed.

In 26 cases of suspected *XK* typhus, blood taken in the early stages (fourth to sixth day) was inoculated into rabbits, 52 rabbits being used. Four of the cases proved to be leptospirosis, and none was later confirmed as *XK* typhus. A non-specific eye

TABLE X.

Titres at which Agglutination was Obtained according to Days of the Disease.

Type.	Titre and Day.
*X*19	—— (7), 1/50 (9), 1/500 (11), 1/1000 (18)
XK	—— (8), 1/125 (15)
XK	—— (4), 1/125 (12), 1/125 (21)
XK	—— (4), 1/500 (13), 1/1000 (20)
XK	—— (4), 1/500 (16)
XK	—— (5), —— (11), 1/1000 (21)
XK	—— (8), 1/2500 (15), 1/125 (22)
XK	—— (4), 1/250 (18)
XK	—— (8), 1/500 (14)
XK	—— (4), —— (11), 1/500 (21)

(NOTE.—Day of illness shown in parentheses after the titre.)

reaction occurred in a number of the rabbits; but no reaction corresponding to the specific reaction was seen, nor were agglutinins for *Bacillus proteus XK* demonstrated by weekly agglutination tests done for six weeks after inoculation.

In four cases of *XK* typhus, in which blood in the early stages was available, reactions in the rabbits' eyes suggestive of the specific reaction were obtained. In none of the rabbits were agglutinins for *Bacillus proteus XK* demonstrated by repeated agglutination tests. Attempts at the passage of the suspected infected aqueous humour did not produce reaction in the eyes of other rabbits, nor were Rickettsia bodies identified in the aqueous humour or in scrapings of Descemet's membrane.

The following are details of the reaction observed in a rabbit inoculated intraocularly with blood taken on the ninth day of sickness (day of death) in a fatal *XK* case. No eschar was present.

The left eye of rabbit I was inoculated with 0·1 cubic centimetre of blood on May 23, 1936. There had been no response to the Weil-Felix test. Up to May 31, 1936, there was slight congestion of the pericorneal vessels. On June 1, 1936, the pericorneal congestion was more pronounced and iritis was present. This progressed, and on June 3, 1936, some clouding of the cornea was evident. On June 4, 1936, there were marked iritis, clouding of the cornea and slight turbidity of the aqueous humour. The iritis was still more severe on June 5, 1936, and aqueous humour was withdrawn and 0·1 cubic centimetre inoculated intraocularly into each of two rabbits (IIa and IIb). On that day the sera of rabbits I, IIa and IIb failed to react. The reaction of rabbit I was about the same on June 6, 1936, and from that day the reaction gradually lessened. On June 8, 1936, rabbit I still had severe iritis, and rabbits IIa and IIb were each reinoculated with 0·1 cubic centimetre of the turbid aqueous humour.

On June 12, 1936, iritis was still present; there were clouding of the cornea and turbidity of the aqueous humour. The pericorneal congestion had practically disappeared. There was still no response to the Weil-Felix test. Rabbits IIa and IIb were further reinoculated, intraocularly. On June 14, 1936, the pericorneal congestion had disappeared. On June 15, 1936, there was still clouding of the cornea, and pannus formation was evident. Iritis was still present but had greatly lessened.

On June 22, 1936, there was still no reaction to the Weil-Felix test, and the eye reaction had practically disappeared, with the exception of residual clouding of the cornea. There was no response to the Weil-Felix test on July 10, 1936, or on August 4, 1936. On the latter date slight cloudiness of the cornea was still present. Neither of the rabbits IIa and IIb showed a suggestive eye reaction, nor was the presence of agglutinins demonstrated.

Rodent Reservoirs and Insect Vectors of the Viruses.

No conclusive evidence in regard to the existence of infection with either the *X*19 or *XK* type of virus in wild rodents has been found. Altogether 104 rodents (91 live rats and 13 live bandicoots) have been examined for agglutinins for *Bacillus proteus X*19 and *Bacillus proteus XK* and combed for ectoparasites. Additional live rodents from the area were examined by Dr. Baldwin and Dr. Sawers and Mr. Taylor, of the School of Public Health and Tropical Medicine, University of Sydney, in September and October, 1935. The identity of the rodents, the localities in which they were trapped, the ectoparasites found, and the highest titres with *Bacillus proteus XK* which were observed, are shown in Table XI.

TABLE XI.

Ectoparasites Found on Rodents.

Locality.	Rodent.	Number.	Ectoparasites.	Agglutination.
City	*Rattus rattus rattus.*	24	Fleas and mites.	1/25 (*K*)
Various parts of country	*Rattus rattus rattus.*	15	Fleas and mites.	
	Hydromys chrysogaster.	3		
	Melomys littoralis.	8	Mites and lice.	1/25 (*K*)
	Perameles macrura.	8	Fleas, mites, ticks.	
	Perameles nasuta.	1	29 ticks.	1/250 (*K*)
Intake area	*Rattus rattus rattus*	19	Fleas and mites.	
	Hydromys chrysogaster.	2	Fleas and mites.	
	Melomys littoralis.	5	Mites.	1/80 (*K*)
	Rattus culmorum	7	Ticks and mites.	
	Perameles macrura.	4	Fleas, ticks, mites.	1/40 (*K*)

The highest titre found in the rodents was 1 in 250 with *Bacillus proteus XK* in a bandicoot (*Perameles nasuta*) from Yarrabah. On this bandicoot 29 ticks (*Ixodes holocyclus*) were found. Inoculation of the crushed ticks and of the bandicoot's brain were made into the eyes of rabbits; but no characteristic eye reactions or agglutinins developed.

Tunica washings, spleen, brain and crushed mites, from rats in which agglutinins for *Bacillus proteus XK* in a titre of 1 in 25 or higher were present, were also inoculated intraocularly into rabbits. No specific reactions were observed.

THE INTRACUTANEOUS TUBERCULIN TEST OF MANTOUX: A METHOD EMPLOYING A SINGLE INJECTION.[1]

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Introduction.

WITHIN the last few years a purified protein derivative of tuberculin has been introduced, a solid, of constant and low molecular weight, non-sensitising on repeated injection, unable to produce focal reactions, stable indefinitely and very highly potent in the production of the skin reaction in animals which have been infected with the tubercle bacillus. This substance is now known as tuberculin *P.P.D.* The pioneer studies of its use for the Mantoux test by E. R. Long, J. D. Aronson and Florence Seibert,[1] and their recommendations as to dosage, first made possible perfect uniformity in tuberculin surveys of populations in different parts of the world. This scheme of dosage has since been generally followed, and is familiar to all who use the Mantoux test.

The Two-Injection Technique.

Long, Aronson and Seibert recommend that two doses of tuberculin *P.P.D.* be employed — an initial intracutaneous injection of 0·00002 milligramme, which detects a high proportion of positive reactors with a minimum of severe reactions, and a second injection, administered to those who have not reacted to the first, of 0·005 milligramme, which has been found to detect all the reactors to the ordinary doses of a highly potent old tuberculin. These doses of tuberculin *P.P.D.* are given dissolved in 0·1 cubic centimetre of buffered saline solution.

The present authors employ this scheme of dosage where practicable. But in the light of experience we suggest that a slightly different criterion from that usually advised be employed in deciding what is a weak reaction to the test if sore arms and blistering are to be avoided. J. D. Aronson[2] has pointed out that sharp reactions are more likely to occur with the first small dose than with the second dose, 250 times as great, after failure to react to the first. But the importance of deciding rightly what is and what is not a failure to react to the first dose is illustrated by the following experience.

The first group of subjects tested by us numbered 72, nurses and masseuses, and their average age was twenty-four years: the average age of the 282 college freshmen and detention scholars whose reactions were the basis of the pioneer survey of Long, Aronson and Seibert for the determination of an optimum dosage, was something less than eighteen years. Whereas none of their subjects, it seems, had severe reactions (though they do not give the dimensions of the reactions), one of our subjects had a vesicular reaction to the first dose, and six had severe reactions to the second and complained of soreness and stiffness in the arm.

One subject, who was classed as a non-reactor to the first dose, but who had nevertheless a minute wheal at the site of injection, after the second dose developed a necrotic area 28 millimetres in diameter, surrounded by an area of erythema 80 millimetres in diameter, and had soreness and stiffness lasting for several days.

Another, who received the second dose after a doubtful reaction to the first, developed a blister 30 millimetres across, surrounded by a red swollen area 120 millimetres in its greatest diameter.

At this time we were following the orthodox criteria,[2][3] and we did not recognize a reaction unless it consisted of an area of œdema at least five millimetres in its greatest diameter. We now believe that it is possible for the experienced observer to be sure of the specificity of smaller reactions down to three millimetres. If such small reactions are disregarded when they occur after the first dose of tuberculin *P.P.D.* and if the second dose is then injected, the reaction to the second dose may be very severe.

The Evolution of a Single-Injection Technique.

The need for a single-injection technique for the Mantoux test has long been recognized. In this country considerable difficulty is being encountered in inducing members of the general population to have tuberculin tests, and it is so difficult at present to arrange to carry out one injection and interpret it two days later that a second injection is usually out of the question.

It was resolved to try to find a dose of tuberculin *P.P.D.* which might be used as a single injection in population surveys to detect a high percentage, if not all, of the total positive reactors with a minimum of untoward reactions. The chief difficulty was expected to be the occasional occurrence of a sharp reaction by a supersensitive subject to a very small dose.

In all previous experiments the requisite dose of tuberculin, whatever its amount, had been given in an injection of 0·1 cubic centimetre. Now 0·1 cubic centimetre is a comparatively large amount of any substance to inject into the skin, and it produces a substantial wheal when injected.

It was felt that a smaller injection ought to produce an allergic response in a susceptible subject as readily recognizable as that produced by a slightly larger injection of the same strength, but that a smaller injection should produce it on a smaller scale, so that a sharp reaction would give rise to less discomfort.

[1] Commenced with the aid of a grant from the Kolling Institute of Medical Research, Royal North Shore Hospital of Sydney, and continued under a grant from the National Health and Medical Research Council of the Commonwealth of Australia.

ILLUSTRATIONS TO THE ARTICLE BY Dr. REGINALD WEBSTER.

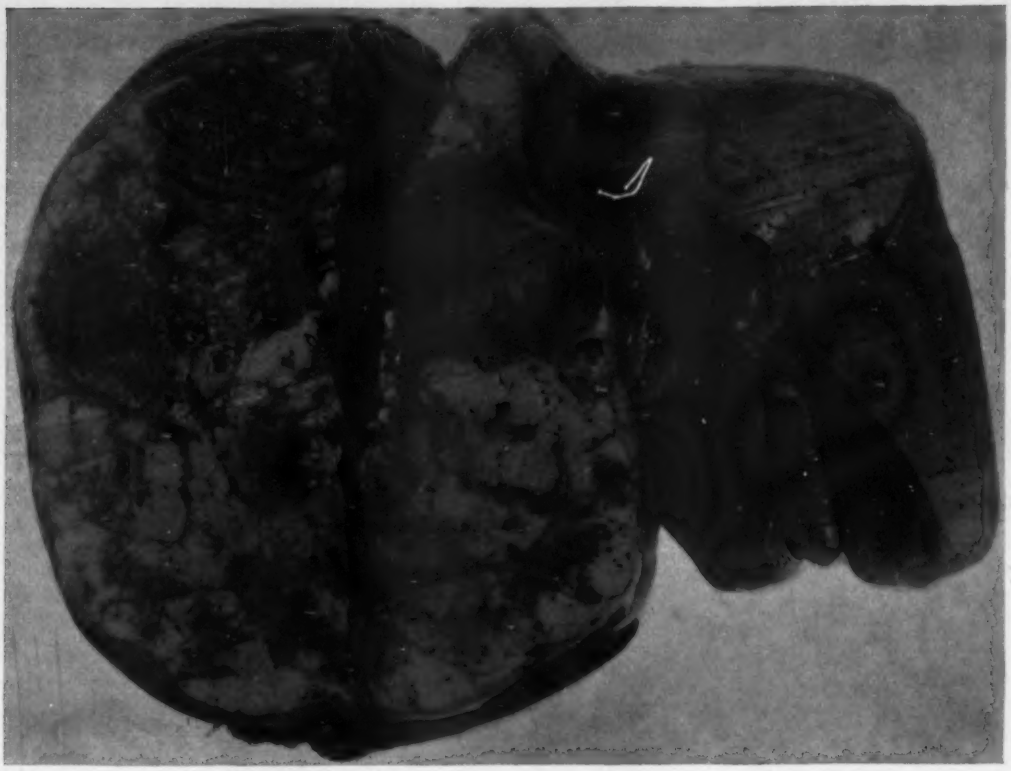

Figure XVI.
Photograph of museum specimen of malignant hepatoma.

Figure XVII.
Low-power photomicrograph of neoplasm, showing a small area of osteoid tissue with calcification.

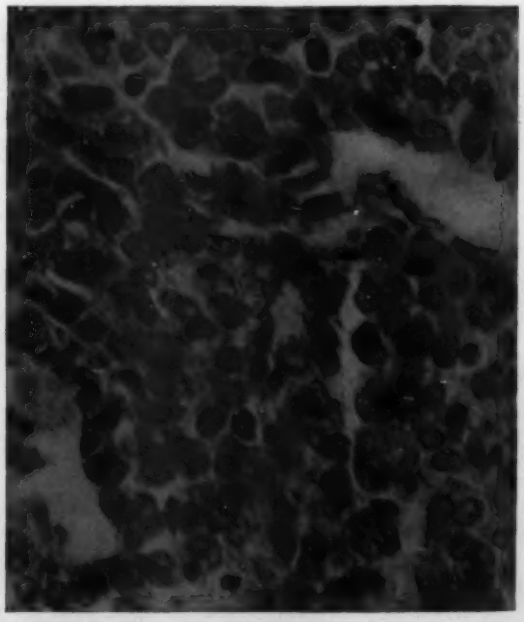

Figure XVIII.
High-power photomicrograph of malignant hepatoma, showing the alveolar arrangement in the neoplastic cells.

ILLUSTRATIONS TO THE ARTICLES BY Dr. E. GRAEME ROBERTSON.

Figure I.
Lateral X ray view of the skull, showing calcification. The two rounded shadows posteriorly are thrown by the chorioid plexuses, in which calcium salts have been deposited.

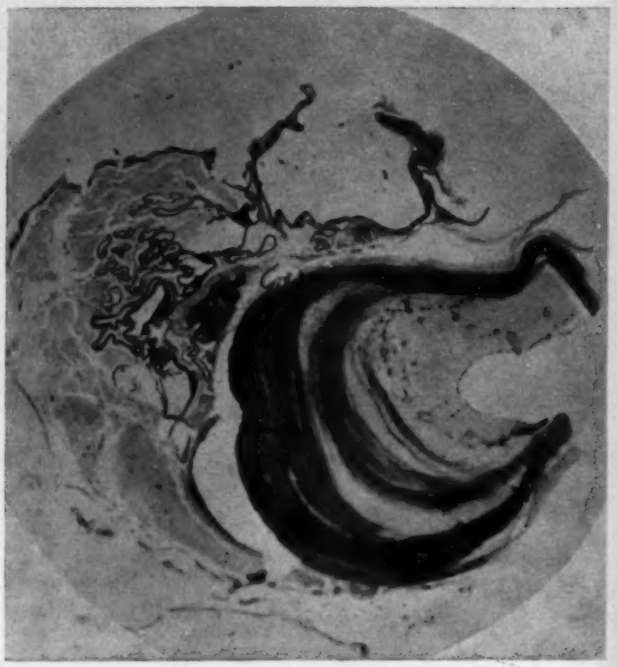

Figure II.
Transverse section of the intradural contents in the lower part of the cervical region (×5). The angioma is seen between the compressed spinal cord and the dense laminated blood clot. The largest vein has ruptured near a point where its wall is much thickened. (Stain: hæmatoxylin and eosin.)

ILLUSTRATION TO THE ARTICLE BY Dr REGINALD WEBSTER.

Figure XIX.
Low-power photomicrograph, showing an extensive tract of osteoid tissue and irregular calcification.

Figure III.
Photomicrograph of the angioma. (Weigert's elastin stain.)

TABLE I.

Dimensions of Reactions to Two Injections of Tuberculin P.P.D. Made at the Same Time.

Number of Subject.	Sex.	Age in Years.	Reaction to Injection of 0·00125 Milligramme of Tuberculin *P.P.D.* in 0·025 Cubic Centimetre of Diluent into the Left Arm.		Reaction to Injection of 0·005 Milligramme of Tuberculin *P.P.D.* in 0·1 Cubic Centimetre of Diluent into the Right Arm.	
			Diameter of Raised Area in Millimetres.	Diameter of Blush in Millimetres.	Diameter of Raised Area in Millimetres.	Diameter of Blush in Millimetres.
1	M.	25	7	10	10	20
2	M.	21	8	18	13	26
3	M.	19	5	12	17	48
4	M.	26	10	25	16	66S[1]
5	M.	37	11	25	17	40
6	M.	29	8	14	16	38
7	M.	25	10	20	17	35
8	M.	10	5	8	9	18
9	M.	31	9	15	11	20
10	M.	30	10	30	13	33
11	M.	24	10	40	20	75VS[1]
12	M.	62	8	18	18	42
13	M.	34	7	20	10	18
14	M.	20	10	20	13	20
15	M.	17	14	45VS	18	70VST[1]
16	M.	26	13	45	18	65
17	M.	66	6	13	7	15
18	M.	28	10	0	15	0
19	M.	51	14	60V	16	65VS
20	M.	57	10	45	14	50
21	M.	37	11	38	13	26
22	M.	26	9	20	18	43
23	M.	—	9	20	15	26
24	M.	—	8	15	14	23
25	M.	—	13	16	13	20
26	M.	—	9	12	7	30
27	M.	—	7	0	10	30
28	M.	—	7	0	13	0
29	M.	—	10	23	17	43
30	M.	—	5	16	10	45
31	M.	—	10	38	12	58VS
32	M.	—	9	0	10	37
33	M.	—	12	0	22	45S
34	F.	33	8	20	18	32
35	F.	28	13	25	18	35
36	F.	22	13	25	14	25
37	F.	29	11	35V	18	60VS
38	F.	28	6	20	9	30
39	F.	31	13	16	25	31
40	F.	15	7	14	20	25
41	F.	17	10	20	15	50
42	F.	34	18	64	18	72
43	F.	30	0	0	0	0
44	F.	52	0	0	0	0
45	F.	54	0	0	0	0
46	F.	21	7	25	10	60
47	F.	37	8	35	10	40
48	M.	41	4	7	5	14
49	M.	15	0	0	0	0
50	M.	49	5	0	5	0
51	M.	32	7	0	9	0
52	M.	26	15	35	17	73
53	M.	53	7	10	9	18
54	F.	43	7	15	12	20
55	F.	64	8	0	15	0
56	F.	66	6	19	10	28
57	F.	43	0	0	0	0
58[4]	F.	53	0	0	0	0
59	F.	30	11	16	11	17
60[5]	F.	16	0	0	3	0
61	M.	17	0	0	0	0
62	M.	38	11	0	16	0
63	M.	28	13	16	12	33
64	M.	24	11	27	15	53
65	F.	32	10	16	14	34
66	F.	28	5	9	10	21
67	F.	21	8	18	16	49
68	F.	27	7	9	8	15
69	F.	23	8	19	10	22
70	F.	19	0	0	0	0
71	F.	50	9	17	12	39
72	F.	62	15	40	18	50
73	F.	18	15	0	18	0
74	F.	25	0	0	0	0
75	F.	34	4	0	8	0
76	F.	46	3	0	3	0
77	F.	24	6	17	8	35
78	F.	23	7	13	13	38
79	F.	24	0	0	0	0
80	F.	31	7	15	9	35
81	F.	39	0	0	0	0
82	F.	20	0	0	0	0
83	F.	56	4	0	7	0
84	F.	27	5	10	6	17
85	F.	25	8	15	10	25
86	F.	38	3	0	4	0

[1] V = vesiculation.
[2] S = sore arm.
[3] T = stiff arm.
[4] Subject Number 58 had a non-specific erythematous reaction ten millimetres in diameter on each arm. She was suffering from congestive heart failure.
[5] Subject Number 60 did not react to the smaller dose. There was a very slight reaction to the larger dose, in our opinion specific, but of such dimensions as to be classed as negative according to orthodox criteria. Subject Number 60 was a girl in hospital for thyreotoxicosis.

TABLE I.—*Continued.*

Dimensions of Reactions to Two Injections of Tuberculin P.P.D. Made at the Same Time.

Number of Subject.	Sex.	Age in Years.	Reaction to Injection of 0·00125 Milligramme of Tuberculin *P.P.D.* in 0·025 Cubic Centimetre of Diluent into the Left Arm.		Reaction to Injection of 0·005 Milligramme of Tuberculin *P.P.D.* in 0·1 Cubic Centimetre of Diluent into the Right Arm.	
			Diameter of Raised Area in Millimetres.	Diameter of Blush in Millimetres.	Diameter of Raised Area in Millimetres.	Diameter of Blush in Millimetres.
87	F.	29	0	0	0	0
88	F.	39	10	0	13	0
89	F.	56	0	0	0	0
90	F.	18	8	25	9	32
91	F.	28	0	0	0	0
92	F.	31	7	13	8	18
93	F.	19	4	0	4	0
94	F.	36	8	12	9	24
95	F.	37	6	0	8	20
96	F.	25	0	0	0	0
97	F.	65	7	0	8	0
98	F.	38	8	0	8	15
99	F.	17	0	0	0	0
100	F.	21	3	0	4	0
101	F.	42	4	0	7	0
102	F.	38	0	0	0	0
103	F.	66	5	0	5	0
104[1]	F.	29	0	0	4	0
105	F.	28	0	0	0	0
106	F.	34	6	0	10	14
107	F.	34	7	15	9	20
108	F.	38	8	0	12	0
109	F.	36	0	0	0	0
110	F.	39	5	0	7	0
111	F.	26	10	15	13	36
112	F.	60	7	0	11	25
113	F.	29	3	0	5	0
114	F.	41	10	15	17	32
115	F.	35	8	25	12	32
116	F.	22	0	0	0	0
117	F.	19	8	0	10	0
118	F.	52	4	0	6	0
119	F.	17	4	0	5	0
120	F.	20	0	0	0	0
121	F.	28	0	0	0	0
122	F.	30	3	0	5	0
123	F.	53	0	0	0	0

[1] Subject Number 104 did not react to the smaller dose. There was a very slight reaction to the larger dose, in our opinion specific, but of such dimensions as to be classed as negative according to orthodox criteria. Subject Number 104 was a woman with puerperal psychosis.

Accordingly 123 volunteers, some known reactors and some clinic patients not previously tested, were given into the right arm an injection of 0·005 milligramme of tuberculin *P.P.D.* in 0·1 cubic centimetre of diluent, and into the left arm an injection of 0·00125 milligramme of tuberculin *P.P.D.* in 0·025 cubic centimetre of diluent, the same dilution but one-quarter the volume. Both injections were given at the same time. It was found to be easy to measure the smaller dose with as much or almost as much accuracy as the larger dose. The results are set out in Table I.

The orthodox second dose of 0·005 milligramme of tuberculin *P.P.D.* is approximately equivalent to 1·0 milligramme (American notation) of old tuberculin[(1)] or to 1·0 cubic centimetre of a 1 : 1,000 dilution. Therefore one-quarter of the dose is yet equivalent in tuberculin to two and a half times the 0·1 cubic centimetre of 1 : 1,000 old tuberculin, used by P. D'A. Hart in his well-known "London Investigation" as the initial injection in the majority of his cases and as the sole injection in many, and stated by him to be in general sufficient for routine purposes.

From this experiment, which is being extended as opportunity offers, it is concluded that an injection of one-quarter of the volume of the standard second strength injection of tuberculin *P.P.D.* detects all or nearly all the reactors to the Mantoux test; that the specificity or otherwise of the smaller reactions is still unmistakable by the experienced interpreter; and that the sharper reactions are of substantially less severity. Hence, though we recommend that the scheme of dosage of Long, Aronson and Seibert should be followed where practicable, if only one injection can be given, this may safely and effectively consist of 0·00125 milligramme of tuberculin *P.P.D.* in 0·025 cubic centimetre of diluent. An experience of over 1,000 such injections has shown that, though sharp reactions are relatively frequent, they are practically never large enough to be inconvenient, and that the proportion of reactors is as great as with the larger injection. Of the first 500 adult subjects tested with the single injection technique (it is not fair to include children because sharp reactions in them are rare), 33 had some degree of vesiculation; of these, the reaction was large (over 17 millimetres of area of œdema) and more or less (usually less) sore in 10; and in five some tenderness was complained of when the reaction was neither large nor vesicular. Only four subjects required treatment, three for blistering and one for itching.

Summary.

The introduction of a purified protein derivative of tuberculin (tuberculin *P.P.D.*) within recent years has made the intracutaneous tuberculin test safer and capable of standardization. The authors review the dosage of tuberculin *P.P.D.* for the intracutaneous test.

In respect of the orthodox two-injection test, new criteria for deciding what constitutes a reaction to the first injection are suggested with a view to the avoidance of severe reactions.

A single-injection technique has been evolved in which the volume of the injection is one-fortieth of a cubic centimetre instead of four times that amount, and which, it is claimed, detects all or nearly all the reactors to the orthodox doses of tuberculin with a minimum of sharp reactions.

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Reports of Cases.

PATHOLOGICAL REPORTS FROM THE CHILDREN'S HOSPITAL, MELBOURNE.

By REGINALD WEBSTER, M.D., D.Sc.,
Pathologist to the Childrens' Hospital, Melbourne.

XII. MALIGNANT (TERATOID) HEPATOMA IN INFANCY.

As a further stage in the process of bringing out interesting specimens from the recesses of the pathological museum of the Children's Hospital, I wish to present an example of malignant hepatoma, which occurred in a baby, aged six months, and was rendered doubly interesting by the teratoid features which it displayed. At the same time opportunity will be taken to place on record two other instances of primary carcinoma of the liver in infancy, less complex in their histological structure in that in neither was there any suggestion of a "mixed tumour".

Malignant disease arising primarily in the liver is rare at all ages, and in the adult would appear to be intimately related to cirrhosis, there being little doubt that the regenerative overgrowth which attempts to compensate for the degenerative parenchymal changes occasioned by cirrhosis and the factors which have brought it about occasionally becomes excessive and neoplastic. The aetiological participation of cirrhosis, however, can be ruled out of the present discussion, which concerns three babies, Harold S., Ronald S. and Douglas D., aged six months, four months and eighteen months respectively.

The clinical history in each instance was a simple story of progressive asthenia associated with the presence of a large tumour, apparent on physical examination, in the right upper quadrant of the abdomen. Only one of the three diminutive patients displayed icterus; in two an exploratory operation was performed only to reveal an apparently inoperable and hopeless condition.

The autopsy on the first child, Harold S., was performed on September 27, 1923, by Dr. Robert Southby, whose notes state that the baby's nutrition had been surprisingly well maintained, and that there was general icterus. The liver was greatly enlarged, the enlargement being confined to the right lobe, the left to all appearances being normal in size, colour and consistency.

Almost the whole of the right lobe of the liver was occupied by a well-defined tumour (Figure XVI), of such a consistency as to give an impression of a cystic character when the organ was palpated prior to section. A section through the liver in a coronal plane showed a generally soft, globular neoplasm, limited by a distinct capsule and subdivided by a number of well-developed fibrous fasciculi. The cross-section of the growth was generally pallid in the lower half, dark and hæmorrhagic in appearance in the upper half. At first sight it appeared that the growth was confined to the right lobe of the liver, but on closer inspection it was seen to have effected invasion of the upper portion of the left lobe also.

It was noteworthy that there were no metastases, either in the immediately related lymphatic glands or in distant organs, particularly the lungs.

In the examination of microscopic sections prepared from tissue selected from different portions of the tumour, I was impressed by the diversity of histological structures which appeared. The tumour was traversed by bands of connective tissue which imparted to it a lobular structure. There were many fields comprised of poorly-staining hepatic parenchyma in an advanced state of fatty degeneration. Contrasting with these were other frequently occurring fields in which masses of hyperchromatic cells, displaying numerous mitotic figures, were arranged in columns and cords in no way resembling the disposition of cells in the normal hepatic lobule (Figure XVII). An attempt at an alveolar arrangement was discernible among these obviously malignant cells (Figure XVIII), which were of epithelial character and seemed clearly to have arisen from the hepatic parenchyma. A remarkable and prominent component of the neoplasm was a peculiarly hyaline osteoid tissue exhibiting foci of unsystematized calcification (Figure XIX). The presence of depots of actively proliferating connective tissue cells, which, if viewed in an isolated high-power field, would spell sarcoma to any microscopist, along with the foregoing characteristics, forced the conclusion that the growth was to be classed as a "mixed tumour".

The microscopic findings in this tumour suggest that if it were subjected to a searching method of analysis, such as that applied by Willis[1] in his study of teratomata, a variety of components sufficient to warrant its description as a true teratoma might be disclosed. At all events, I recollect leaving the study of the sections impressed with their general histological resemblance to Wilms's embryoma of the kidney, and rightly or wrongly have been accustomed to regard this tumour as an hepatic analogue of Wilms's tumour.

The *post mortem* examination of the second of these infants, Ronald S., was conducted on October 1, 1922, by Dr. H. Boyd Graham, who recorded the presence of an exceptionally large hepatic tumour. The liver occupied the upper half of the abdomen and extended to a point just below the umbilicus. The capsule of the organ was intact, the gall-bladder and bile-ducts were in normal position and contained bile, but with the exception of the Spigelian lobe and the lip of the left lobe, the whole of the liver, to quote Dr. Graham's description, was "converted into a vascular encephaloid mass".

Although the condition could be regarded as nothing other than a malignant neoplasm, no metastases could be found in a systematic search of the organs of the thorax and abdomen.

The third of these children, Douglas D., aged eighteen months, died on August 12, 1925. At the *post mortem* examination I found a large neoplastic mass in the right lobe of the liver. No hepatic tissue was to be distinguished in the right lobe, the surface of which was rendered nodular by the protrusion of lobules of the malignant growth. There was no obvious hæmorrhage, necrosis or degenerative change in the neoplastic tissue, which was on the whole very vascular and of soft consistence. The

glands in the portal fissure and along the superior border of the pancreas were enlarged and involved in the malignant growth; but there was no dissemination elsewhere. There was no jaundice nor ascites, and the viscera, apart from the liver and the immediately related lymphatic glands, showed no pathological changes.

The strong suggestion of malignancy in the hepatic neoplasms in both of these children was abundantly confirmed by examination of microscopic sections.

Sections of the liver of the child, Ronald S., showed sheets of proliferating cells which, though they had lost all trace of normal hepatic design, were not so anaplastic as to render their origin from the liver parenchymal cell unrecognizable. They were supported by a minimum of stroma, at times assumed a tubular arrangement, and rioted among a profusion of thin-walled blood vessels.

The tumour in the case of the child Douglas D. was of similar nature, distinctly epithelial in origin, although not so vascular and rather more differentiated than that of Ronald S. The component cells presented varying formations, tubules, columns and masses, with an alveolar effect where tubular columns had been cut tangentially. In neither of the two neoplasms were any of the teratoid characters described as occurring in the first one of the group in evidence.

Having regard to the rarity of newgrowths arising primarily in the liver, whether in children or adults, the pathologist who makes the autopsy in a case of apparent primary hepatic neoplasm should be careful to determine that the tumour in the liver does not represent a metastasis from a malignant growth which has arisen elsewhere. In children a neuroblastoma referable to a small primary growth in the adrenal is the most likely source of error. Holt[6] records the occurrence of a neuroblastoma in the liver of a child six weeks of age, and states that a tumour of the same nature in the liver of a patient no older than eighteen days has been reported.

Even in children that fertile source of secondary deposits in the liver, carcinoma in the gastro-intestinal tract, cannot be left entirely out of calculation; for in the pathological museum at the Children's Hospital is a specimen of carcinoma of the sigmoid colon which occurred in a girl, aged ten years.

With respect to the three tumours which I have presented as primary malignant hepatic neoplasms in infancy, I can answer for it that with the exception of the immediately related lymphatic glands, none of the other organs of the abdomen, or of the thorax, was implicated in any form of tumour growth in the baby on whom I performed the autopsy. Dr. Graham and Dr. Southby careful observers both, state specifically in their *post mortem* records that no pathological changes were to be observed in the viscera apart from the liver.

Thus in two of these children no metastases were present, and in the third metastatic deposit was limited to the regional lymphatic glands. The question of metastasis in primary malignant disease of the liver in general, apart from that peculiar to infancy, is one of some interest. According to Ewing, the tendency to metastasis in malignant hepatoma is not great, and extrahepatic metastases occur more frequently and much earlier in biliary cancer than in malignant hepatoma.

O. L. Castle,[3] in a review of primary carcinoma of the liver in childhood, tabulates 42 reported cases in which the ages of the patients ranged from one day to sixteen years. Metastases are given as present in nine and absent in ten; but no less than twenty-three are listed as unclassified with respect to metastases. This is an unsatisfactory feature of Castle's table, for which, of course, the author is in no way responsible. It was published in 1914, and has been criticized on other grounds by Kilfoy and Terry,[4] who, in reporting a case of primary carcinoma of the liver in childhood in 1929, published a table which they presented as "showing all cases reported to date". The table was a reproduction of that of Castle, with the addition of two cases, the authors' own and that of Wollstein and Mixsell.[5] From this it would appear that only two cases were reported in the sixteen-year period 1914 to 1929, Kilfoy and Terry taking no cognizance of the report of a case of primary carcinoma of the liver in childhood by J. P. Crozer Griffith,[9] in 1918, that author's sifting of the literature, and his conclusion that 58 of the reported cases merited recognition as having been satisfactorily substantiated. This is the more extraordinary as the reference to Griffith's article is included in the bibliography of the subject compiled by Kilfoy and Terry.

The criticism of Castle's table advanced by Kilfoy and Terry is that in sixteen of the cases which comprise it the diagnosis is doubtful. In fifteen others they regard the case reports as unsatisfactory, being deficient with respect to autopsy findings or confirmatory microscopic evidence. In only thirteen of the forty-four did they consider all requirements met, namely, adequate clinical history, detailed autopsy findings and convincing reports on microscopic sections.

It is extremely difficult to assess precisely how many genuine examples of malignant hepatoma in childhood have been recorded. The reports upon which existing estimates have been made date from as early as 1841 (Olivier), and it is probable that many of the earlier records would not survive scrutiny by present-day histological standards.

Rolleston and McNee[7] state that of sixty-five cases of reputed primary carcinoma of the liver, thirty-two occurred in children aged under ten years.

Although figures are scarcely necessary to gain acceptance for the statement that malignant hepatoma is rare, it is perhaps of interest to note that this condition has been uncovered three times in the 4,711 autopsies of which records exist at the Children's Hospital. These records cover a period of twenty-seven years, and it is a curious circumstance that the three examples of malignant hepatoma in infancy here reported with chapter and verse as to autopsy findings and microscopic confirmation, occurred within the three years 1922 to 1925. The autopsy records show no instance before 1922, and there has been none since 1925.

Three interesting and completely attested case reports, published since 1929, are those of F. W. McRae[13] (1935), T. W. McCreary[8] (1937) and E. Norman Jamieson[10] (1935). In that of F. W. McRae[13] the pathological aspects are presented in accurate detail, and it was extremely interesting to read of indubitable evidence in the microscopic sections of teratoid features similar to those which I have endeavoured to describe in the tumour from the child Harold S. The meticulous pathological description is summarized in the conclusion that the growth is a congenital mixed tumour, primarily adenoma and adenocarcinoma, with areas of osteoid tissue, atypical blood vessels, and a small area of keratinized epithelium providing evidence of an embryonic origin.

In a search for other examples of teratoid components in hepatic neoplasms of infancy and childhood, I found that Castle[3] mentions an instance of primary liver carcinoma in an infant, aged seven months, as reported by Idzumi. In this tumour was found teratoid tissue. Yamagiwa has also reported a primary teratoid adenocarcinoma in a girl one year old, but I have not been able to gain access to the original reports of these Japanese writers.

As long ago as 1908 Philipp[11] described as a "primary, epithelial, destructive and therefore carcinomatous mixed tumour, containing traces of cartilage", a growth the size of a small apple which he had obtained at autopsy on a male infant aged nine months.

McRae's patient was a male infant, six months of age, on whom an exploratory operation was performed and the removal of a large hepatic tumour undertaken. The infant died on the fifth day after the operation, but death was considered to be due in no way to hæmorrhage from the liver and was attributed to hepatic insufficiency and partial intestinal obstruction occasioned by adhesions which had formed around two gauze packs. McRae expresses himself as of opinion that surgical removal should always be attempted, and that recurrence is not greatly to be feared. The relative freedom from metastasis has already been noted, but I feel sure that if

F. W. McRae could see the specimens I have discussed he would modify his opinion at least to the extent of qualifying the word "always".

In commenting upon McRae's patient, Dr. A. O. Singleton quoted the case of a child, then three years of age and apparently healthy, from whom a large tumour of the liver, pronounced subsequently by Dr. Ewing to be an adenocarcinoma, had been removed by cautery at the age of seventeen days. The tumour weighed 175 grammes (six ounces) and the baby six pounds.

A malignant hepatoma of the liver was also removed from Castle's patient, an infant, aged ten and a half months. A favourable circumstance was the fact that the tumour was pedunculated. Surgical convalescence was very satisfactory until the fourteenth day, when what appeared to be intercurrent gastro-enteritis determined the child's death. Necropsy was not permitted, although "zealously urged".

The child discussed by T. W. McCreary[9] provided an interesting observation on the mode of dissemination of liver carcinoma. The clinical picture of an acute abdominal crisis precipitated a laparotomy which disclosed intraperitoneal hæmorrhage, traceable to the rupture of a tumour on the inferior surface of the right lobe of the liver. Biopsy showed a primary carcinoma of the liver of hepatoma type, a diagnosis which was later confirmed by Dr. James Ewing. Six weeks later the child died, and at autopsy there were found multiple nodular masses protruding from the surface of the liver, contrasting with the single tumour present at the time of operation. In spite of the multiple extensions into the liver, there were no metastases in other viscera or in neighbouring lymph nodes. This observation would appear to bear out Ewing's teaching, that the cells of a hepatoma invade capillaries early, causing intravascular thrombi, which are incapable of surviving transplantation from the liver, but may initiate secondary nodules in the liver.

E. Norman Jamieson[10] reports the case of a boy, aged three and a half years, in whom an exploratory operation revealed a single massive tumour of the liver. The photomicrographs are very convincing in support of the diagnosis of malignant hepatoma. No metastases were discovered in an autopsy limited to the abdominal viscera.

Those wishing to pursue the subject of primary carcinoma of the liver in childhood further will find a good bibliography attached to the article by Kilfoy and Terry.[4]

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A CASE OF CALCIFIED CEREBRAL TUMOUR.[1]

By E. GRAEME ROBERTSON, M.D., M.R.C.P.,
Melbourne.

N.R., A TRAINED NURSE, aged forty-eight years, gave a queer episodic history when admitted to the Royal Melbourne Hospital under the care of Dr. H. F. Maudsley. Four years before, when she was on night duty, a colleague had commented upon a defect of speech of which the patient had been unaware. She began and finished a sentence, but left out words in the middle, although she had mentally formulated the words. She felt quite well and was able to write the night report; but when she read it, the matron commented upon the fact that she was leaving out words. She was told that her mouth was crooked; but she was unable to see this in a mirror. The disability lasted for thirty-six hours only. After this attack she felt constantly tired and was easily exhausted; but she was otherwise well until nine months before her admission to hospital, when for several days her right arm felt weak. This weakness was so slight that, she said, if she had not had the other arm to compare it with she would not have noticed it. Six months later, when attempting to get up from bed after doing two weeks' night duty, she vomited copiously and suffered frontal headache for a few hours. After this she was so exhausted that she was unable to leave her bed for several days. In the three weeks before admission to hospital she had three attacks of unconsciousness, of short duration and unaccompanied by muscular twitching. In the slightest of these she slowly subsided to the floor "like a deflating balloon" and things went black for only a second. During these few weeks she suffered from slight vague headache on top of her head, lasting only for brief periods.

On examination, commencing papilloedema, with striate hæmorrhages, was noticed in the left fundus. The visual fields were full; there were slight signs on the right side of involvement of the pyramidal tract, and sensation of all forms was normal. The cerebro-spinal fluid was under a pressure of 130 millimetres of water, and jugular compression gave a normal response. Apart from a high protein content (75 milligrammes per 100 cubic centimetres) the cerebro-spinal fluid was normal. There was no reaction to the Casoni test. X ray examination of the skull revealed a faintly calcified irregularly globular shadow, with its medial border situated 0·6 centimetre (one-quarter of an inch) to the left of the mid-line, above and posterior to the *sella turcica*, and rising 5·0 centimetres (two inches) above the latter (Figure I).

The patient became more and more drowsy and the hemiplegia increased, so that fourteen days later she was almost comatose. In the next fourteen days drowsiness deepened into coma and the hemiparesis became hemiplegia.

On July 16, 1937, Dr. A. E. Coates explored the left hemisphere around the Sylvian point, and by extending the bone flap inspected the frontal lobe, the lesser wing of the sphenoid, and the suprasellar region. No lesion was found. A brain exploring needle was passed deeply into the brain, but failed to reveal evidence of abnormality. The wound was closed and a decompression established.

She was transferred to the Austin Hospital six weeks after operation. At this stage there was a gross hemiparesis and the patient led a vegetable-like existence, incontinent, and, when roused to a sufficient degree, giving evidence of a gross jargon aphasia. So she remained for two months, and then a progressive improvement commenced in her mental and physical state, until at the present time she appears to be a normal woman, if somewhat loquacious. Complete neurological examination fails to reveal an abnormal sign, although, measured with the dynamometer, the strength of the right hand is slightly less than the left.

[1] The patient in this case was shown at a meeting of the Victorian Branch of the British Medical Association at the Austin Hospital on April 20, 1938.

The interest of the case became greater when the period of unconsciousness was discussed with the patient. She remembered nothing after her first day in the Royal Melbourne Hospital, "as if I had gone to sleep and awakened". During this long period, however, she was living in a life of phantasy, so vivid as to seem real in retrospect. She states definitely that she seemed to live through a number of experiences in this time, and is able to state the order in which they occurred. Some of these "novels", as she calls them, were connected with episodes in her past life, and might have been descriptions of everyday events. One consists in a description in great detail of the daily events on a voyage to Tahiti and San Francisco, on which she has actually been. The shortest and most fantastic "story" contains much material of interest to the psychologist and is perhaps worth recording.

> I die. My shoes and stockings are taken off me, and I am fully clothed otherwise. I am placed in a coffin and then I am taken away to a Catholic church in Camberwell and a memorial service is held.
>
> After that I am removed, not outside the church, but into an adjoining room, about twelve by fourteen feet, and the coffin is placed on the floor. Then a voice speaks and says: "You had better come to bed." I put my hands up and the lid of the coffin simply goes up before me.
>
> I push it away, sit up and look round, and the room is quite bare, with the exception of three bunks on one side, one above the other. The voice had come from one of these bunks, and without answering I simply get out of my coffin and into the bottom bunk. Almost at once I seem to be in a sound sleep.
>
> Once again a voice speaks. This time it says "Follow me", so without answering I step out of the bunk, and I notice this other person does not make for the door, but instead she walks across the room to a corner and in the floor there is a trapdoor. She opens this up and she descends. I follow and we go down some steps. When we come to the bottom we come to a corridor which is perfectly dark. We walk along, descending all the time. This seems to be quite a long way, and then at the end of it we come to daylight again. We walk out into open space.
>
> I find that I am in an enormous graveyard. I just stand and look around and see the beautiful monuments and statues, such as I have never seen before. So I walk by myself in my bare feet and stand in front of a tomb and read down the tombstones. Then I pass on to the next and do the same, one after the other. Some I notice are 300 years old, some less, and right down to the present day. I continue this walk, up one path and down another, never getting tired, reading and sometimes dusting off the dirt, tidying the flowers and so on.
>
> Later on the day seems to be ending and I walk right down into a hollow like a valley. As I do so, I notice there are others besides myself, and eventually I get down to a beautiful big pond surrounded by rushes and bamboos. On the water there are water-lillies of three different shades, pale pink, pale blue and white.
>
> Sitting round the edge there are four other figures, not taking any notice of one another, just sitting in a restful attitude. So I sit down too. It is very restful, and then night seems to come and I go to sleep.
>
> When dawn comes we all rise and stroll off on our walks, just separately, and I do the same as I did the previous day: go around looking at the figures and tombs and dusting them and removing the dead flowers and walking on and on.
>
> Then I see a funeral coming in, with horses being driven. I sit down quite near, unobserved by any of the people. I watch the burial taking place. The same as the day before, I go down to the pond again. Each day follows the same until evidently quite a long time has passed, and this time I see a funeral coming in again. I watch everything, and at the end of it everyone except one gentleman returns to the mourning coaches. This one walks over to me, as if he has seen me, and he says: "How many marks have you got?" and I reply: "98 sir". He says: "I think that is very good; now would you like to go back to the world?" and I reply: "Yes sir". So he says: "Well, you follow me."
>
> And this is the first time I have spoken. They went up a hill. I have never walked up that hill before, and I follow on. They leave me behind because I am only walking in my bare feet. When I am about half way up the hill they disappear out of a big open gate. So I just go on walking slowly, and at last I come up and pass through the big space and stop, and then a big revolving gate comes round and closes and I am back in the world again.

The only association with her previous life is that she visited a number of graveyards when touring around the world. Another story recounts being kidnapped and after a long voyage being transformed into a mermaid by an ingenious incorporation within a fish.

Speculation could be endless as to the meaning of these symptoms and the implications of this vivid fantastic life, with withdrawal from reality, apparently due to a structural cerebral lesion. The nature of the lesion and its exact situation are also uncertain. It is possible that it is an ependymoma growing in the frontal horn of the left lateral ventricle. In an attempt to elucidate this, air was introduced into the lumbar theca; but it failed to enter the ventricles, a fact which may be significant. The recovery must be due to the decompression allowing restitution of the circulation of cerebro-spinal fluid and the subsidence of œdema. She is so well that she desires no further investigations.

A CASE OF ARTERIAL ANGIOMA OF THE SPINAL CORD.[1]

By E. GRAEME ROBERTSON, M.D., M.R.C.P.,
Melbourne.

M.W., A GIRL, aged sixteen years, gave the following history.

Three years before admission to the Austin Hospital she had suddenly felt giddy and the next day had stayed in bed. In the evening she had felt pain in the back of the neck and down the right arm, and within two hours the right arm and leg had become powerless and the whole of her right side, below the neck, had become numb. She was unable to feel pinching or pin-pricks. Two weeks later she gradually lost the use of her left arm and leg, and was unable to pass urine and fæces. Three months later the weakness in the left arm increased, and three months afterwards she had difficulty in breathing, due apparently to an increase in the weakness of the intercostal muscles or to involvement of the phrenic nerve. The retention of urine gave place to incontinence and, later, precipitant evacuation after taking purgatives was very troublesome. The weakness of all four limbs prevented any useful movement, but showed a tendency to improvement; the numbness became less marked.

Examination revealed quadriplegia, with wasting and contractures, mixed signs of upper and lower motor neurone lesions in the right upper limb, and signs of an upper motor neurone lesion in the other limbs. There was but little sensory loss, although there was a reduction of superficial sensation below the fourth cervical dermatome. While the patient was in hospital a further increase in the paralysis occurred on two occasions. She eventually died, abruptly, of respiratory failure, fifteen months after admission.

It is interesting to note that she was sent to the Austin Hospital with a diagnosis of poliomyelitis. The

[1] The patient in this case was shown at a meeting of the Victorian Branch of the British Medical Association at the Austin Hospital on April 20, 1938.

cerebro-spinal fluid contained no cells, the protein content was increased, and the colloidal gold curve was 4445555555. The response of the cerebro-spinal fluid pressure to jugular compression was reported to be normal, although this was probably incorrect. At autopsy the spinal cord, the base of the pons, the cerebellum and the interpeduncular region were covered by blood, subarachnoid in location. The lower three cervical segments of the spinal cord were reduced to a crescentic remnant surmounted on one side by a large cap of dense material of white cartilaginous appearance (Figure II). Microscopically this was seen to be laminated blood clot. Between the spinal cord and the organized fibrin, which were both included within a covering of thickened pia-arachnoid, there was a reddish area which contained a plexus of vessels, apparently a racemose arterial aneurysm (Figure III). The vessels were of varying size and had thick muscular walls containing elastin. In places there was intimal proliferation. There was a definite rupture of the thin wall of the largest vessel near an area where aneurysmal dilatation was commencing, and was apparently the source of a terminal hæmorrhage. Earlier hæmorrhages into the spinal cord, or interference with its blood supply, probably accounted for the abrupt onset of the initial symptoms and for the intermittent increase in the damage. A tendency to improvement in between these bouts was apparent in spite of the tumour-like appearance of the laminated blood clot.

A CASE OF CEREBRAL ADHESIONS.

By LEONARD L. SNOW, M.B., Ch.M.,
Inverell, New South Wales.

C.R., AGED fourteen years, was brought to me from western Queensland on January 15, 1936, on account of increasingly frequent epileptic fits.

At birth an instrumental delivery damaged the right side of the vault of his skull. Convulsive fits occurred so frequently and so severely that his life was despaired of. However, they gradually lessened in frequency and ceased at about the age of two years.

He had no more fits until the age of thirteen. In the next twelve months he had eighteen fits, five of which occurred in the last three months before his visit to me. At the commencement of a fit there would be a peculiar feeling in his left cheek, which would be paralysed for five or ten minutes; he would froth at the mouth and the froth would become bloody; after struggling for five or ten minutes he would lie deeply unconscious for an hour, and after that have a peaceful sleep for up to half an hour. On waking he would be "as good as ever" and have no headache.

The patient had a frightened semi-imbecile look, and was anxious about the likely occurrence of another fit. He was given "Luminal", with slight improvement.

On March 17, 1937, he was suffering from acute prolonged headaches associated with antral disease. X ray examination revealed opacity of the left antrum and slight opacity of the right antrum.

On March 18, 1937, under a general anæsthetic, a radical left antrum operation was performed. He left hospital on March 24, 1937. His headaches vanished and his general condition improved; but the fits now came much more frequently. Taking 0·3 gramme (five grains) of "Luminal" a day, he was having five or six fits in twenty-four hours; so on April 7, 1937, his mother agreed to an X ray examination of the vault of the skull in detail. Radiography revealed three areas of thinning in the right frontal bone (where, his mother now informed me, the birth injuries had first shown).

On May 1, 1937, his mother brought him again, as he stated that life was no good to him, and he begged for an operation if possible, as he would rather die in or after operation than live as he was.

On May 2, 1937, at 9 p.m., he was given 0·18 gramme (three grains) of "Hebaral Sodium" in a capsule. On May 3, 1937, at 6 a.m., he was given two capsules, each containing 0·18 gramme (three grains) of "Hebaral Sodium", and at 7 a.m. one more such capsule. At 8 a.m. operation was performed with the assistance of Dr. Dent, of Inverell. Local infiltration with a 0·5% solution of "Novocain" with adrenaline was used, and a squarish osteoplastic flap was raised, to embrace the three areas of thinning of the right frontal bone. The operation took forty-five minutes.

When an incision was made through the *dura mater* three firm round adhesions, each about 1·6 centimetres (five-eighths of an inch) long, were observed running from the dura to the frontal lobe of the brain. When the first adhesion was drawn on, the patient stated that a fit was about to commence, starting in the left ankle. When the second adhesion was drawn on, the patient stated that a fit was about to commence, starting in the left shoulder. When the third adhesion was drawn on, the patient stated that a fit was about to commence, starting in the left cheek, where all his fits had started.

The three adhesions were removed, and an oblong of "Cedco" rubber dam, 5·0 by 3·75 centimetres (two by one and a half inches) was placed between the *dura mater* and arachnoid.

Before operation, taking 0·3 gramme (five grains) of "Luminal" a day, he had five or six fits daily. After the operation, though taking no "Luminal", he had no fit for about sixty hours, when he had a major fit. The next day he had a minor fit, and he has had none since.

He was last seen by me on April 19, 1938 (eleven and a half months after operation). The most striking thing about him now was his thoroughly bright and intelligent appearance. He had had no more fits and had completely lost his look of terror. He was now able to live a normal lad's life, playing tennis and attending the cinema—two things he had not dared to do before operation.

Reviews.

PULMONARY CARCINOMA.

"PRIMARY CARCINOMA OF THE LUNG", a book by Edwin J. Simons, M.D., provides us with a thorough study of a subject which is demanding more attention on account of its increased incidence and the difficulty in its diagnosis.[1] Dr. J. Arthur Myers in a foreword states that Dr. Simon, who is a general practitioner in a rural district, corrects the erroneous belief that the physician must have access to a complete library and an extensive laboratory and every other facility found in a large hospital or medical school, before he is able to make a valuable contribution to medical knowledge.

The book is a compact manual of 263 pages and contains an extensive bibliography. The illustrations include two bronchoscopic views in colour of bronchial carcinoma and adenoma. After a short introduction the author discusses the incidence of the disease. Statistics of most of the countries of the world show an absolute relative increase in lung cancer, particularly since the War. It is not generally recognized that 10% of all cancers are located in the lungs. Although the ætiology is unknown, the author arrives at the conclusion that chronic irritation, whether it be chemical, mechanical, bacterial, radio-active or thermal, is the real activating or causative factor. Under the heading of pathology he discusses the evidence for inclusion of the oat cell tumours among the pulmonary carcinomata.

Röntgenological considerations are discussed at length, and many X ray pictures are presented to illustrate the

[1] "Primary Carcinoma of the Lung", by E. J. Simons, M.D.; 1937. Chicago: The Year Book Publishers Incorporated. Medium 8vo, pp. 263, with illustrations. Price: $5·00 net.

text. The statement that the differential diagnosis cannot be made from the X ray appearances alone seems very true; the clinical history is of equal importance. The author believes that the more general use of the bronchoscope is responsible for the earlier histological diagnosis of these lesions, the indication for its use being clinical or radiological evidence of bronchial obstruction. The endoscopist will readily appreciate the force of these statements.

The opinions of different authorities for and against radiotherapy are given, and the technique of this mode of treatment is clearly described. The more recent surgical procedures, lobectomy and pneumonectomy, are discussed. Graham and Singer's work is quoted and the operative technique of W. F. Rientroff described. The author considers that wide surgical excision has more successes to its credit than any other mode of treatment.

This compact manual is a valuable compendium of knowledge relating to primary carcinoma of the lung. It will be found of value to the general practitioner, general surgeon, endoscopist and radiologist.

A DIET HANDBOOK.

As Lord Dawson of Penn points out in a short but laudatory foreword, Miss Rose Simmonds has fulfilled a great need by writing her "Handbook of Diets".[1] Every medical practitioner, whether he specializes or is in general practice, has perforce to be dietitian as well as doctor. Unless diets are given item by item, patients seldom follow them with the necessary care; but they will carry out detailed written directions. The well-planned and simply prepared schedules and diet sheets here set out will be a boon to the doctor who has little time to elaborate them himself.

The lists of food values and of standard weights and heights are taken from reliable sources and save reference to several volumes. Miss Simmonds's experience as dietitian to the London Hospital and to the Hammersmith Hospital Post-Graduate School has well fitted her to speak on the practical value of diets. She keeps in mind the purse of the patient as well as the palatability of the food, and offers alternatives and progressive schedules to avoid monotony.

Since it is the British custom to provide four meals a day—breakfast, dinner, tea and supper—it seems a little difficult at first sight to adapt English dietaries to the Australian hospital practice of giving three major meals and at least three in-between snacks or "teas". This difficulty may be surmounted, however, if the fluids and light articles provided at "tea" in the handbook are retained for morning and afternoon tea, and the more solid articles included in the midday dinner. The diets calculated to supply ample minerals and the "reducing" diets will be found of particular value. The sections devoted to the nutrition of the expectant and nursing mother and of the infant and young child are also worthy of close perusal.

CARDIAC FAILURE.

THOSE who have studied East and Bain's work on cardiology in the "Recent Advances" series will show no hesitation in reading "Failure of the Heart and Circulation", by Terence East.[2] This pocket monograph explains in an easy and lucid manner many of the problems concerned. It is full of facts helpful to both student and practitioner.

After discussing the mechanism of cardiac failure, he proceeds to enlarge on its effects, first on the circulatory system and secondly on the other organs. He includes the gist of Harrison's work on the cause of dyspnœa. Comparing the clinical syndromes of right and left ventricular failure, he emphasizes the importance of recognition of the early signs of failure. The part played by thyreotoxicosis, pregnancy and vaso-vagal disturbance in the causation of circulatory failure is discussed. Failure of the coronary circulation and peripheral circulatory failure are also considered.

Treatment is discussed at some length, due stress being placed on the urgent necessity for its application at an early stage in failure. The author's advocacy of the injection of adrenaline for Stokes-Adams attacks might well have been tempered by the warning that it is first necessary to be sure that they are due to ventricular standstill and not to ventricular fibrillation, for in the latter case the result of the injection might be disastrous.

He issues a warning that the intramuscular injection of "Salyrgan" may cause necrosis; but this complication must be very rare. In spite of its being slightly less effective by this route, the drug is still very serviceable and a boon to stout patients with inaccessible veins.

Pathologists who have had the opportunity of studying a large series of hearts from patients who have died in typical attacks of coronary occlusion, will disagree with his statement that "in the large majority a thrombus forms . . . and blocks the vessel".

In spite of these debatable points, the little book is readable and helpful, and not a mere collection of facts.

Notes on Books, Current Journals and New Appliances.

MATERIA MEDICA FOR NURSES.

THERE is apparently a demand for Professor A. Muir Crawford's book on *materia medica* for nurses, as four editions have been necessary in ten years.[1] The information it contains is sound, and student nurses will find much here that will be of value to them; but there are certain defects. The arrangement is unsatisfactory, for both study and reference. Some important substances receive no mention; for example: neither carbon tetrachloride nor oil of chenopodium finds a place among the anthelmintics. The book has the merit of brevity and no doubt will help many nurses to pass their examinations.

WOMEN'S HEALTH AND HAPPINESS.

THE ideal book on education in matters of sex has yet to be written. "Cyrus Fullerton", in his "Happiness and Health in Womanhood",[2] gives a simple and readable description of the reproductive process, but does not quite succeed in avoiding mawkishness, and dwells rather long on the grisly results of promiscuity. The book has been written as a "guide to better understanding and closer friendship between mother and daughter". It will be of value to some women and girls, but of possible danger to others.

[1] "Handbook of Diets", by R. M. Simmonds, S.R.N., with a foreword by Lord Dawson of Penn, P.C., G.C.V.O., K.C.B., M.D.; Second Edition; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 171. Price: 7s. 6d. net.

[2] "Pocket Monographs on Practical Medicine. Failure of the Heart and Circulation", by T. East, M.A., D.M., F.R.C.P.; 1937. London: John Bale, Sons and Curnow Limited. Foolscap 8vo, pp. 138. Price: 2s. 6d. net.

[1] "Materia Medica for Nurses", by A. M. Crawford, M.D., F.R.F.P.S.G.; Fourth Edition; 1937. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 114. Price: 3s. 6d. net.

[2] "Happiness and Health in Womanhood", by "Cyrus Fullerton", with a foreword by H. Sutton, O.B.E., M.D., Ch.B., B.Sc., D.P.H., E. S. Morris, M.D., Ch.M., D.P.H., and J. G. Drew, M.A., M.B., B.Ch., M.R.C.S., L.R.C.P., D.P.H., D.T.M., D.T.H., F.R.S.I.; 1937. Sydney: Father and Son Welfare Movement. Crown 8vo, pp. 74, with illustrations. Price: 2s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 3, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

DIET.

ONE of the most striking developments in practical medicine during recent years has been the increasing attention devoted to problems of diet. In each large hospital nowadays a dietitian is employed as a permanent member of the staff and presides over a kitchen in which special meals of known composition are prepared. The feeding of patients is no longer the haphazard affair it once was. The medical student in former years was given no instruction whatever in dietetics. In hospital practice it was usual for attending physicians or surgeons to order, by rule of thumb, either milk and milk foods, light diet or full diet for the patients under their care. No attempt was made to prescribe a properly balanced diet, and even in cases of diabetes the estimation of the protein, fat and carbohydrate elements in the patients' meals was almost a problem for a consultant. Beyond warning sufferers from nephritis against the dangers of eating too much meat, the clinician had little advice to offer on any question bearing on the relation between food and physical well-being.

Those rough and ready days are over, and there is now a tendency to swing in the opposite direction, towards an accuracy that is in some ways extreme. There are hospitals, notably in the United States of America, in which the medical officers habitually prescribe their patients' diets in terms of so many grammes of protein, carbohydrate and fat; then the hospital dietitian, after a solemn conference with the patient as to his whimsies concerning food, elaborates a diet containing the necessary number of Calories. The work of feeding diabetics and nephritics is more complicated still, and is the task of a special staff working in the diet kitchen, many of whom are students proceeding to a diploma in dietetics. The weighing and preparation of special diets for patients suffering from certain forms of illness require consummate skill, but instances have been cited in which a salt-free meal intended for a patient suffering from nephritis has been defeated of its purpose because a carefree nurse has placed a salt-cellar beside the vase of violets upon the patient's tray.

The times are past when medical men believed that our bodily needs would be satisfied if only our food were digestible, palatable and of sufficient amount; and the great cause of the change has been the recognition of the fact, even by the common man, that the prime cause of a number of distressing but quite avoidable diseases is a lack of accessory food factors, the existence of which was previously unsuspected, or the want of a number of inorganic salts, the importance of which had previously been unknown.

Such is the attention paid to problems of diet today that during the past year or two the annual publications on some aspect or other of food requirements have exceeded five thousand, all of them intended for the use of scientific workers. To quote but one instance, there has recently appeared a book ("Food Tables",[1] by V. H. Mottram and

[1] "Food Tables", by V. H. Mottram, M.A., and E. M. Radloff, B.Sc., Ph.D.; 1937. London: Edward Arnold and Company. Demy 8vo, pp. 63. Price: 5s. net.

Ellen M. Radloff) which consists of sixty-two pages of closely printed type and in which the composition of all foods in common use has been tabulated in alphabetical order.

Another remarkable change in our attitude towards food is the manner in which books on the subject, written by high authorities and intended for the layman, pour from the presses of the world. One such book is "Your Diet and Your Health", from the pen of Dr. Morris Fishbein,[1] known to all medical men as the editor of *The Journal of the American Medical Association* and of the magazine *Hygeia*. In this little volume are discussed, in language which any intelligent reader can follow, such questions as the cost of foods and foodstuffs, the diets suitable to all ages and conditions, and the nonsensical fads and cults which have been foisted upon the public from time to time. Few medical men realize, until they read such a publication as this, how many charlatans have taken it upon themselves, usually at great profit, to prescribe menus for their fellows. Many of us remember the cult of Fletcherization, which nearly killed Henry James, and the Graham system, which forbade the taking of meat, sauces, tea, coffee, alcohol, pepper and mustard, and condemned its miserable followers to a diet of vegetables, nuts, salt and water, the latter preferably pure. Then there was the savant, Alexander Haig, who announced in the nineties that uric acid was the great factor in the causation of disease. The evil acid, said Haig, is the result of eating meat, and he who swallows meat is a candidate for high blood pressure, rheumatism, diabetes, gout, nephritis, headache, epilepsy, anæmia and mental depression. It took scientific medicine a good twenty years to disprove these alarming contentions.

There are few interested in bodily economy who do not know something, at least by hearsay, of the diet advocated by Dr. Hay and his associates, though it should be said that the Hay diets do not, as some imagine, involve the swallowing of hay and other cereals. The central pillar of Dr. Hay's dietetic temple is the belief that proteins, carbohydrates and fats cannot be successfully digested if eaten together, and that continued attempts at such a dietetic crime may result in acidosis and a lingering death. It is within the knowledge of all students of clinical medicine and of physiology that Dr. Hay's ideas have no foundation, and Dr. Fishbein has performed a valuable public service in laying before the public the evidence which refutes them.

As Professor E. W. H. Cruickshank observes in his book "Food and Physical Fitness",[1] it is impossible for anybody to set down a standard diet or even a series of standard diets suitable for universal use. There is no one protein, carbohydrate or fat of which the equivalent cannot be found in more foods than one. The ordinary housewife should have a general knowledge of dietetic requirements; amongst the public there is a widespread demand, too long neglected, for information of this kind. Some Australian physician, interested in the science of dietetics, would do a service to the community by writing a manual upon the subject.

[1] "Your Diet and Your Health", by M. Fishbein, M.D.; 1937. New York: McGraw-Hill Book Company Incorporated; Australia: Angus and Robertson Limited. Large crown 8vo, pp. 308, with illustrations. Price: 15s. net.

Current Comment.

CARBON DIOXIDE AND ITS EMPLOYMENT.

THE use of carbon dioxide in many conditions has become a habit and is not always guided by clear and logical thinking. From time to time the occurrence of perplexing convulsions in patients under ether anæsthesia has been reported and many and various are the explanations suggested. R. M. Waters has very pronounced views on the matter.[2] He quotes from an article written by K. B. Pinson in 1927. Pinson regarded these convulsions largely as an outcome of the action of carbon dioxide on patients sensitive to it by nature or by reason of toxæmia, pyrexia and so forth while they are under the influence of ether, which itself has a conspicuous effect on breathing. He considered that an essential in treatment was the prompt riddance of excess of carbon dioxide from the body. He further wrote

[1] "Food and Physical Fitness", by E. W. H. Cruickshank, M.D., D.Sc., M.R.C.P., F.R.S.E., with a foreword by J. B. Orr, D.S.O., M.D., D.Sc., F.R.S.; 1938. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 160. Price: 5s. net.

[2] *The Canadian Medical Association Journal*, March, 1938.

that in his opinion the administration of carbon dioxide would aggravate the condition. Waters states his conviction that, had Pinson's observations been more carefully considered, much futile discussion concerning convulsions under anæsthesia would have been avoided. He remarks that the trigger effect of an excess of carbon dioxide in the production of convulsions under certain exceptional circumstances is only one of its dangerous properties, and that, as we continue every year to meet patients with more severe biochemical and physiological imbalance, it becomes increasingly necessary to prevent the slightest derangement of normal physiological conditions. Waters is emphatic that carbon dioxide is greatly overrated as a therapeutic agent, and that, as a final insult to previously deranged function, it not infrequently determines death. He considers that clinical misapprehensions concerning the physiology of carbon dioxide had their inception in the work of Yandell Henderson, who deduced from his investigations that surgical shock resulted from a low concentration of carbon dioxide in the blood and tissues. He also considered that low values of carbon dioxide in the blood and tissues were a usual accompaniment of anæsthesia and that hyperventilation of the lungs effected by the use of carbon dioxide and oxygen should prevent post-operative pulmonary atelectasis. Waters holds that all of these deductions are fallacious. He further states that the belief that shock depends entirely on a low concentration of carbon dioxide is disproved by the fact that M. H. Seevers and his associates "hyperventilated" dogs for many hours, reducing the blood carbon dioxide to points below fifteen volumes *per centum* without extreme drops in blood pressure and without evidence of shock. Carbon dioxide added to the inspired air of men and animals whose blood pressures were low as a result of profound spinal anæsthesia has in some cases resulted in a further fall of blood pressure. It never resulted in a more sustained rise in pressure than could be produced by ventilation of the lungs with oxygen alone.

Eastman and Kreiselman demonstrated in their attempts to reestablish respiration after an oxygen crisis or a complete apnœa due to oxygen want that carbon dioxide, added to the oxygen used in artificial respiration, caused in some instances a further fall in blood pressure, and always gave a less satisfactory circulatory recovery than that following artificial respiration with oxygen alone. Waters admits that Henderson's assumption that anæsthesia is accompanied by a low concentration of carbon dioxide in the alveoli, blood and tissues may have been justified during the past century, but he maintains that it is not so now. Preoperative medication with certain drugs results in a diminution of minute-volume respiration, less efficient ventilation and loading of carbon dioxide in the alveoli, blood and tissues. Many inhalation agents tend in the first instance to stimulate respiration, but their effects rarely or never suffice to overcome the high concentration of carbon dioxide which is present at the commencement of induction and which has been caused by premedicant agents. Waters asserts that clinical experience does not substantiate the contention that mixtures of carbon dioxide and oxygen diminish the incidence of atelectasis. Isolated lobes of a lung with intact circulation can be demonstrated to absorb gases at varying rates, with the formation of varying degrees of atelectasis according to the receptivity of the blood for their transport. K. E. Lemmer and E. A. Rovenstine have published observations showing the comparative rates of absorption to "moderate collapse" following inflation of an isolated lobe and complete obstruction of its bronchus. Waters, however, points out that the figures do not indicate satisfactorily the fact that lobes filled with the rapidly absorbed gases, when studied for greater periods, become completely atelectatic, ultimately resembling liver in aspect. Such lobes, when filled with nitrogen, never resemble liver in consistency.

Waters demands that the administration of carbon dioxide be conducted with a complete appreciation of its physiology. It should be remembered that carbon dioxide is a waste product of body metabolism in the same way as are urinary constituents. Reduction in the efficiency of respiration tends to dam back carbon dioxide into the tissues. It should also be remembered that all drugs employed to relieve pain have a tendency to interfere with the normal function of pulmonary ventilation. Non-volatile agents do so on account of their tendency to depress respiration. Volatile agents produce this effect through the mechanical difficulties of administering them without obstruction of respiration. When it is intended to add carbon dioxide to the inspired air, the legitimacy of such measures should be called in question. Waters holds that, with extremely rare exceptions, artificial respiration without carbon dioxide would serve the patient much better. The extreme variability of its effect on different individuals and on the same individual at different times must never be forgotten. Carbon dioxide, in concentrations greater than 10%, is not compatible with consciousness in normal persons. Waters states that carbon dioxide is never used at Wisconsin to prevent or cure surgical shock. During anæsthesia carbon dioxide is seldom or never employed. The anæsthetist uses every effort to avoid the formation of dead spaces at the upper respiratory tract and to prevent rebreathing. Carbon dioxide mixed with oxygen is never used to secure post-anæsthetic hyperventilation. The induction of active hyperpnœa, which leaves in the more remote alveolar spaces a rapidly and completely absorbable medium, such as oxygen, is considered by Waters and his associates to be productive of more harm than benefit. Whenever the employment of carbon dioxide is followed by muscular twitching or by depressed and irregular breathing, it must be forthwith discontinued.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Dermatitis.

RUPERT HALLAM (*The Lancet*, June 4, 1938) describes the causation and treatment of dermatitis. A low-grade resistance of the skin to physical irritants is present. "Mental resistance" may also play a part, for scratching or rubbing due to lack of self-control may change a trivial localized patch of inflammation into a generalized dermatitis, which may cripple the patient for many months. The author describes the causes and treatment of traumatic dermatitis, which he considers to be the inflammatory reaction of the skin to the application of an external irritant. In sensitization dermatitis the clinical appearances and histological changes are similar in many respects to the reaction which follows trauma, but the mechanism differs. In explanation of the pathology of the reaction, the experiment performed on himself in 1924 by Cranston Low is described. He rubbed a crushed primula leaf into the abraded skin of his forearm. No reaction followed. Three weeks later he again rubbed a leaf into the same site, and on this occasion an intense reaction followed within twelve hours. Before commencing the experiment he could handle the primula with impunity. His skin, once sensitized, has remained susceptible. A traumatic dermatitis is limited to the area of skin which has been in contact with the particular irritant; but a sensitization dermatitis is inclined to spread and even involve distant skin surfaces. The relationship between eczema and sensitization dermatitis is discussed. The third division of the subject is atopic dermatitis, a term applied to infantile eczema and its sequelæ, flexural eczema and prurigo. The author observes that the only scientific treatment of a dermatitis is to try to classify it under the appropriate type. A discriminating history is an essential prelude to diagnosis.

Impetigo Contagiosa.

PAUL TACHAU (*The British Journal of Dermatology and Syphilis*, March, 1938) relates his experiences with *impetigo contagiosa* in Chicago. Whilst in practice in Germany he had seen impetigo of the streptococcal type much more often than the staphylococcal form, which was rare. In Chicago, in 60% to 70% of cases the lesion was of the staphylococcal variety. The relationship of bacteriological findings to clinical type in more than twenty cases corresponded exactly with the experience of Dohi, J. Jadassohn, Lewandowsky, Fuchs and others, in that the bullous type was caused by the staphylococcus. It is important to take cultures from fresh intact blebs. Epstein recently confirmed the findings of Radcliffe-Crocker, Engman, Clegg and others, that the bullous type of impetigo was caused by staphylococci. This was done by inoculation with a pure culture of staphylococcus, a confirmatory culture being taken from the blebs produced. Distinct clinical differences between streptococcal and staphylococcal impetigo are noticeable at first sight. In staphylococcal impetigo the blebs are much larger, more persistent, and remain intact for a few days. Usually they have no areola or the areola is very small. Formation of annular circinate lesions is common. Scabs arising from drying or rupture of the bullæ are thin and serous, like varnish. After the disease has been cured, pigmented spots may be found. Streptococcal impetigo is characterized by pin-point vesicles in the centre of a red spot; but these are seldom seen, because they burst very soon, leaving broad red areolæ and thick, honey-like crusts, which are yellowish or hæmorrhagic, and adherent. When the lesions are healed the red or bluish spots remaining disappear in from three to five weeks. In Europe the streptococcal form is the commoner, but from time to time epidemics of staphylococcal impetigo appear.

Ehlers-Danlos Syndrome.

M. BOLAM (*The British Journal of Dermatology and Syphilis*, April, 1938) describes a case of the Ehlers-Danlos syndrome. The patient was a boy, aged twelve years. The main features of the syndrome, namely, clear elastic skin, hyperextension of the joints and extreme fragility of the skin and blood vessels, were very pronounced. The angiomata and hæmatomata arising in connexion with them probably correspond to the pseudo-tumours described by other authors. An interesting feature of this case was the febrile disturbances which followed the spontaneous appearance of hæmatoma. Headache occurred, with a rise of temperature to 37·8° C. (100° F.), and the patient had to retire to bed. Whenever the skin was broken it took a long time to heal; these lesions frequently became infected. Fatty tumours developed just beneath the skin of the forearm and along the lateral margin of each tibia. Biopsy showed them to be lobules of fat compressed tightly within a fibrous capsule. There were myxomatous changes in the connective tissue, with a particularly loose arrangement of this part of the dermis and an almost complete absence of subcutaneous fat. Protection from injury appears to be the only useful treatment for this congenital error in skin development.

Studies in Nail Growth.

W. E. LE GROS CLARK AND L. H. D. BUXTON (*The British Journal of Dermatology and Syphilis*, May, 1938) state that available information regarding the rate of growth of nails rests on few and unreliable data. The authors' interest in nail growth arose in connexion with problems of nutrition and tissue growth in general. They were concerned with the establishment of the normal variations of nail growth as a standard for comparison of nail growth in various types of nutritional deficiency. Their investigation showed that there was a regular relationship between the growth of the nails of various digits. The growth of the nail on the third digit is consistently greater than that of the others, while the little finger nail is consistently the least rapid in its growth. The rate of growth of the thumb-nail is probably slightly less than that of the nails of the index and ring fingers. The habit of nail-biting is associated with a great acceleration in the growth of the finger nails. Between the ages of ten and twenty-five years there is no variation of nail growth with age. The difference in the rate of nail growth between the left and the right hands is negligible. No conclusive evidence of a sexual difference in the rate of nail growth was obtained. The growth was greater in the summer than in the spring.

Angioma.

GEORGE C. ANDREWS (*Archives of Dermatology and Syphilology*, April 1938) publishes a discussion of the treatment of angioma, based on his observation of 408 patients with vascular nævi, treated at the Vanderbilt Clinic, New York. Of these, 319 were instances of hæmangioma, including both strawberry marks and hæmangioma of the cavernous type, and eight were instances of *lymphangioma circumscriptum*. Two hundred and two of the patients were one year of age or younger. There were multiple lesions in 55 cases and single lesions in 353. Treatment by the injection of sclerosing agents was given in 52 cases. The best results were obtained with quinine and ethyl carbamate. The author considers this the best treatment for hæmangiomata about the eyes and on the lips, and for extensive lesions on the sides of the face or on the hands or wrists. It is usually the best treatment for relatively radio-resistant hæmangiomata, whatever their position, and for *lymphangioma circumscriptum*. The author believes that treatment by injection should be employed more extensively than it is at present, because of its effectiveness, its freedom from danger and its simplicity. Radium therapy was used in 146 cases, either by external application or by interstitial implantation. There were pronounced individual variations in the radio-sensitivity of hæmangiomata. Treatment with radium, in this author's opinion, should be limited to angiomata which are particularly radio-sensitive, and should not be used near the eye or on extensive lesions over bony prominences. Surgical excision was performed in 35 cases. Refrigeration is mentioned as an old method of treatment of established

value. It is useful for small lesions on areas where cosmetic results are not of great importance. It results in superficial necrosis that heals with a white pliable scar.

UROLOGY.

Necrosis of the Testicle.

H. N. DORMAN (*Urologic and Cutaneous Review*, May, 1938) describes eight cases of necrosis of the testicle which occurred in his own practice during a brief period of time. He is of the opinion that the condition is much more common than a study of the literature would indicate. Necrosis is characterized by the sudden onset of pain, a rise in temperature, anorexia, nausea and abdominal distension. The pain is particularly severe and persistent, resisting all measures for its relief. Occlusion of the spermatic artery is believed to be the cause of necrosis of the testicle. This may be due to external pressure (for example, a tight suspensory bandage) or internal pressure, such as torsion, infarct or inflammation around the spermatic artery. As the entire testicular substance will be destroyed, orchidectomy with vasectomy as far as the inguinal ring is recommended as the most expeditious method of treatment. In five cases necrosis of the testicle followed perurethral resection of the bladder neck. In one case this condition followed second-stage prostatectomy, and in another case suprapubic cystotomy and lithotomy.

Hydatid Cyst of the Kidney.

F. BILGER AND P. BRANZEU (*Journal d'Urologie*, December, 1937) describe in detail a case of hydatid disease of the kidney, an affection which they consider rare. The authors declare that the treatment of such cysts should, if possible, be conservative, for nephrectomy in such cases is an operation of a degree of gravity out of all proportion to the benignity of the disease. The mortality of nephrectomy in hydatid disease of the kidney they find from extensive studies to be about 18%. On the other hand, the mortality in cases of hydatid cysts of the kidney in which operation is not performed is only 5%. Moreover, spontaneous cures are not unknown. The oldest of the conservative operations is marsupialization. It has a 5% mortality. The fistula persists as a rule for one to three months. This type of operation is indicated in suppurating cysts and in large cysts with irregular walls. Partial nephrectomy has been advocated by some surgeons, particularly for small cysts which are not very adherent and have only a small area of renal implantation; also for calcified cysts not opening into the interior of the kidney. However, the same advantages can be obtained by practising reduction of the cyst without drainage, after formolization. The cyst is punctured, a 1% solution of formalin is injected, the cyst is then incised and the membrane and daughter cysts are removed. The cyst is then sutured without drainage. The ends of the suture should be attached to the skin surface. Relapse after formolization is exceptional. If fluid collects in the pouch the authors state that a few aspirations will suffice for a cure. This method of treatment should not be practised if infection is present.

Epididymo-Vasostomy.

THE technique of the operation of epididymo-vasostomy is given in detail by M. D. F. Blasuci (*Urologic and Cutaneous Review*, June, 1938). He has devised several innovations, amongst which are the introduction of silver wire sutures with a hypodermic needle and the use of a serum needle and its stylet, specially machined, as a cannula and obturator for the *vas deferens*. The bilateral operation is expected to take two hours, including the microscopic examination of the seminal fluid from the epididymis and the confirmation of the patency of the *vas deferens* above the site of operation. Great stress is laid upon complete control of hæmorrhage without ligatures or clamps. Five successful cases are recorded.

Ureteric Calculi Treated by Indwelling Catheters.

R. L. DOUBMASHKIN AND A. A. SOLOMON (*Urologic and Cutaneous Review*, June, 1938) have analysed the histories of 1,125 patients with ureteric calculus. Their observations lead them to the following conclusions: (i) Indwelling catheters did not produce sufficient dilatation of the ureter to induce the passage of stones measuring six millimetres or more in width. (ii) Acute renal infection followed the use of indwelling catheters in 11% of cases in which there was no previous infection of the urinary tract. For this reason the procedure was abandoned in the absence of infection. (iii) When stone impaction coexisted with acute renal infection, indwelling catheters brought about a rapid amelioration of symptoms and abatement of the infection. (iv) The use of a 5% glucose solution by continuous intravenous drip greatly facilitated the drainage of urine through the catheter. (v) The high proportion of successes in cystoscopic removal of large-sized stones in this series (84%) appeared to be due to ample dilatation of the ureter below the stone with metal dilators and rubber bags. In 257 cases involving 940 manipulations with these instruments there were only three instances of infection.

Radiolucent Vesical Calculi.

K. L. SCHOBER (*Zeitschrift für Urologie*, January, 1938) publishes a new method of skiagraphically demonstrating bladder stones which are radiolucent, especially when for some reason the cystoscope cannot be introduced. Even when this instrument can be passed, a radiographic representation of the stone is of practical value in disclosing the shape and size of the entire stone, which can perhaps be seen only partially in each cystoscopic field. The latter consideration applies even with radio-opaque calculi, so that skiagraphy should always be used, even though successful cystography is available. The principle of the new method is that both the stone and a small amount of injected contrast medium tend to lie on the floor of the bladder when the patient is in a supine position, with the radio-opaque solution surrounding the stone, which forms a filling defect in the contrast medium. The latter is in turn surrounded by a relatively large amount of injected air, the effect of which is to show up the contour and even the details of the surface of the stone or stones in a very clear way. The bladder is emptied through a small Tiemann catheter and ten cubic centimetres of "Abrodil" are injected. Some air is then injected into the bladder with a 150 cubic centimetre syringe until the patient feels the first sensation of pressure. The catheter is then withdrawn and the X ray pictures are taken.

Urinary Calculus.

DRAWING on his own experience and an exhaustive review of the literature, L. D. Keyser (*Bulletin of the New York Academy of Medicine*, February, 1938) states the present position of knowledge of calculi in the urinary tract. The various theories of stone formation are enunciated and commented upon. With these as a basis the treatment of calculus disease is discussed in detail. Special attention is paid to the prevention of recurrence after surgical removal. The article concludes with a system of tables which set out all the procedures recommended for investigation and analysis of these conditions, and the operative, medicinal and dietetic treatment of patients in whom they occur.

Treatment of Pyogenic Cystitis.

C. L. DEMING (*New England Journal of Medicine*, December 30, 1937) believes that pyogenic cystitis has received little scientific consideration. The bladder has definite defence mechanisms against infection. The avenues of access of organisms are few. When a bladder infection persists for more than ten days there is usually an obstruction to the outlet or an internal source of infection. Prophylactic treatment consists in avoiding catheterization as far as possible; when it is unavoidable a soft catheter should be used and the bladder washed out afterwards. The author states that daily irrigations with solutions at a temperature of 48·5° C. for from ten to twenty days will cure many chronic cases. He advises procumbent urination for patients whose infections are due to cystocele or paralysis, and considers that the adoption of this as a daily practice is a good method of preventing further infections.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on May 18, 1938, at the Royal Melbourne Hospital. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff.

Carcinoma of the Sigmoid Colon.

DR. W. A. HAILES showed a man, aged sixty years, who had been admitted to hospital on April 3, 1938, with a history of a sensation of weight in the left iliac fossa of twelve months' duration, and of the passage of bright blood *per rectum* during a period of three months.

X ray examination after a barium enema on March 7, 1938, revealed no obstruction, but very irregular filling at the recto-sigmoid junction. This was thought to be due to a carcinoma. Some negative shadows in the area, it was thought, might be due to polypi.

The patient was an old man, not cachectic. Left facial paresis was present. The abdomen was not tender or rigid, and no mass or viscus could be felt abdominally or *per rectum*. A hernia through the lateral abdominal wall was present on the right side. The hæmoglobin value was 84%.

On April 5, 1938, colostomy of the transverse colon was performed, in order to place at rest the lower portion of the colon and rectum. A mass was felt in the sigmoid colon, capable of resection later. After the operation the patient contracted gastroenteritis with vomiting and frequent passage of fæces and much mucus from the colostomy. The condition ultimately subsided. The distal half of the colon, from the colostomy to the anus, was irrigated daily.

On May 6, 1938, the growth was removed and an end-to-end anastomosis was performed. Some free fluid was present in the abdominal cavity, thought to be the result of the gastroenteritis. It was possible to divide the bowel on the pelvic floor 3·75 centimetres (one and a half inches) below the growth. Microscopic section of the growth showed it to be an adenocarcinoma; the glands were also found to be infiltrated.

Dr. Hailes said that the case illustrated the advantages to be gained, in an operable carcinoma of the left side of the colon, from Devine's method of preventing the colon from functioning. These were an empty and clean colon that could be handled easily and sutured with little risk, because it was not functioning and because nothing was passing along it. The risk of peritonitis, local abscess formation or separation of the anastomosis was reduced to a minimum.

Sarcoma of the Maxilla.

Dr. Hailes's second patient was a man, aged twenty-three years, on whose face a plastic operation had been performed. In 1930 he had presented himself at the hospital with swelling of the left side of the face. A malignant growth was shown by X ray examination; it involved both antra and the nasopharynx. Swelling and redness of the cheek and of the anterior pillar of the tonsil were present, with ulceration inside the cheek, outside the last molar tooth.

The patient had several courses of radium and deep X ray therapy. On October 30, 1934, an operation was performed through the left nasal passage and antrum, which left him with a large opening into the left nasal passage. At this operation a tumour the size of a small orange was removed; it was encapsulated and attached to the posterior edge of the bony septum. Radium needles were inserted.

On his admission to hospital on March 13, 1938, the skin of the left side of the face was scarred and atrophic, the nose was deviated to the left, and a large sinus, 1·9 centimetres by 2·5 centimetres (three-quarters of an inch by one inch) opened into the left nasal cavity, which was bounded medially by an intact septum. There were degenerative changes in the cornea of the left eye. There was no clinical or radiological evidence of recurrence. The margin of the bone deficiency was covered by scar tissue, the result of the radium treatment.

On March 18, 1938, a tube was formed from the skin of the neck for use as a pedicle, a piece of skin 3·75 centimetres (one and a half inches) wide being used for the purpose. On March 29, 1938, two pieces of costal cartilage were inserted under the skin at the right extremity of the future pedicle. At this stage it was intended that this skin should line the inside of the opening. On April 11, 1938, it was decided to turn in flaps from the edge of the opening to line the inside of the opening and to use the pedicle graft for the outside. In the meantime the circulation in the pedicle was stimulated from left to right by a rubber ring tied on the right extremity for one hour each day.

On April 19, 1938, flaps from either side and above and below the opening were turned in and sutured with catgut, the knots being inside the nose. The pedicle, with a flap of skin at its right extremity, was then placed over the opening to cover the outside. The cartilage grafts were found to be unnecessary and were dispensed with. On April 10, 1938, the pedicle was removed. It bled when severed, indicating a good blood supply through the graft itself.

Dr. Hailes took the opportunity of thanking Dr. Fay Maclure for his help in the case.

Duodenal Ulcer.

Dr. Hailes's third patient was a man, aged forty years, who had been admitted to hospital on April 12, 1938. His history was not typical of a duodenal ulcer. For six years he had had attacks of pain and vomiting; the vomiting relieved the pain. He had had eight or nine attacks, each lasting about one week. His last attack had lasted for about five or six months. The pain was a gnawing pain, not related to meals. It began below the umbilicus and moved into the left lower part of the abdomen. It occurred chiefly at night, and was not relieved by powder, but was relieved by vomiting. This relief by vomiting seemed to be the chief feature of the pain. In the few months prior to the meeting he had been vomiting food on the average twice a night and once a day, and had recognized food taken several meals previously. He also complained of flatulence. He stated that five years previously he had vomited blood. He had lost 6·4 kilograms (one stone) in weight in eight months.

On examination he was slightly tender to deep pressure to the right of the umbilicus. Examination after a test meal revealed a large amount of free hydrochloric acid. Examination after a barium meal showed half of the meal still in the stomach after six hours. There was great irregularity of the duodenal cap, which was very irritable. A diagnosis of chronic duodenal ulcer with obstructions was made.

On April 29, 1938, Dr. Hailes performed the Finsterer modification of the Pólya gastrectomy. A chronic duodenal ulcer and some narrowing of the pylorus were seen at operation.

Dr. Hailes said that the case was of interest for several reasons. The first reason was the unusual history, with vomiting at the height of the pain and relief resulting from vomiting. This was unusual in duodenal ulcer. The second reason was the high acidity present in spite of pyloric obstruction. This made the case unsuitable for gastro-enterostomy. The need for partial gastrectomy in order to reduce the acidity and lessen the risk of gastro-jejunal ulceration was also unusual.

Severed Tendons of the Little Finger.

The next patient shown by Dr. Hailes was a young man, aged nineteen years, who had been admitted to hospital on June 10, 1937. Both *flexor profundus* and *flexor sublimis* tendons of the little finger of the left hand had been severed by a penetrating wound. Division

was not complete at the time, but fourteen days later the finger under strain became useless and could not be flexed voluntarily at either interphalangeal joint and the joints were flail-like. The penetration had occurred over the proximal phalanx.

The tendon sheath was opened and the tendons were found to be severed completely where the sublimis split and surrounded the profundus. The *flexor sublimis* tendon was cut away completely, the *flexor profundus* alone being sutured with linen. The hand was dressed with the finger flexed, and movement was commenced in two days.

At the time of the meeting there was full voluntary flexion in the finger, so that the distal phalanx could meet the palm of the hand. Full extension was slightly limited because of a little cicatricial contraction in the scar. Dr. Hailes said that the achievement of almost full function after suture of the profundus tendon alone was of interest, in view of the poor results obtained by suture of the divided slips of the sublimis tendon around the sutured profundus tendon, as was usually advised. The adhesions formed between the two tendons appeared to splint the finger.

Gastric Ulcer.

The next patient shown by Dr. Hailes was a man, aged fifty-one years, who had been admitted to hospital on March 6, 1938, for operation for left inguinal hernia. He appeared very pale, and it was suspected that some more serious condition was present. He complained of vague pain in the left lower part of the abdomen, of eighteen months' duration, and also of pain in the back, of six to nine months' duration. Lately he had felt very fatigued and weak, and had been vomiting food and dark material at variable times, usually within one hour, after meals. He had lost 6·35 kilograms (one stone) in weight in nine months.

An X ray examination of the urinary tract revealed no abnormality. The blood serum gave no response to the Wassermann test. The only definite physical sign on examination of the abdomen was some tenderness in the left hypochondrium.

A blood count done on March 7, 1938, revealed that the hæmoglobin value was 35%. The erythrocytes numbered 2,320,000 and the leucocytes 7,800 per cubic millimetre. The colour index was 0·76. Carcinoma of the stomach was suspected, but examination after a test meal revealed considerable amounts of free hydrochloric acid. No bile, mucus or lactic acid was present, but blood was present.

Examination after a barium meal revealed a large atonic stomach with almost complete pyloric obstruction. No details were seen. After forty-eight hours there was still a large gastric residue.

On March 15, 1938, the abdomen was opened after 683 cubic centimetres (one pint) of blood had been given by transfusion. A very large gastric ulcer was seen in the region of the pylorus. The stomach was adherent to the left lobe of the liver. A gastro-enterostomy was performed. At the time of operation the patient had much occult blood in his stools. His hæmoglobin value was 40% after the transfusion. He was given a course of iron medication, after which his hæmoglobin value slowly improved and the occult blood disappeared from his stools. On April 25, 1938, the hæmoglobin value was 82%.

On March 31, 1938, he was given a barium meal. Examination revealed that the bulk of the meal passed through the stoma; a trace went through the duodenum, which was deformed. A large penetrating ulcer was seen on the posterior surface of the lesser curvature, nearer the fundus than the pylorus. The stoma was at the most dependent part of the gastric curvature.

The patient's hernia was repaired on May 10, 1938. He was very ill; and a Red Cross blood donor could not be used on account of the hospital rule, since he was considered to have carcinoma. The operation had to be performed at an awkward period, shortly after a rigor due to a transfusion from the only donor available. At operation the large ulcer on the lesser curvature did not seem to be the cause of the pyloric obstruction; it was not possible to determine whether there was another ulcer at the pylorus on the posterior surface.

Dr. Hailes remarked that at the time of the meeting the position unfortunately was that there was an unhealed ulcer on the lesser curvature, associated with a reasonably high acidity and a gastro-enterostomy. The patient was very well and was being watched clinically and radiographically for the fate of the ulcer and for any indication of gastric or jejunal ulceration. The procedure carried out was all that was possible at the time; but it was unlikely that this would be the end of the patient's gastric history.

Traumatic Osteoarthritis of the Ankle for Arthrodesis.

Dr. Hailes's last patient was a man, aged thirty-five years. Twelve years earlier he had fractured his right tibia. Afterwards he was able to walk quite well till seven years prior to the meeting, when the ankle swelled and was very painful. He was twice subjected to operation for fixation of the joint, but he still had slight movement.

An X ray examination on September 9, 1937, revealed no bony ankylosis, but an irregularly deformed articular surface at the ankle joint, with adjacent sclerosis. A separate fragment was present anteriorly between the tibia and the talus.

On November 23, 1937, Dr. Hailes excised the scar on the medial side of the ankle. The joint was disorganized. The remains of cartilage and opposed bone surfaces were chiselled off the tibia and talus. A longitudinal incision was made on the outer side of the ankle, the lateral malleolus was chiselled off and the remainder of the joint was excised. Two ivory bone pegs were hammered in. The leg was placed in plaster.

On February 2, 1938, the patient was discharged to the out-patient department, wearing a plaster and using crutches. An X ray examination on March 7, 1938, revealed that union was taking place posteriorly, but none was taking place in the anterior half of the joint. Dr. Hailes remarked that the case was of interest in that it illustrated how difficult it was in some joints to obtain true bony ankylosis. The patient worked as a motor mechanic for five years with fibrous ankylosis. A bad wrench tore some adhesions, and from then on it was impossible for him to walk any distance or to work. Six months after excision of the scar he was free from pain and returned to work.

Mitral Stenosis and Auricular Fibrillation.

DR. L. E. ROTHSTADT showed two patients on behalf of DR. L. E. HURLEY. The first was a man, aged forty-five years. At the age of fifteen years he had had "growing pains", and five years later he had been told that his heart was diseased. Until two years prior to the meeting there had been no symptoms except occasional palpitations, which had not been present since. At that time he developed congestive heart failure with normal rhythm. He was in bed for one week, and after a further week of rest he returned to work. He was free from symptoms until two months before his admission to hospital, when he became short of breath and vomited several times, and soon his ankles became swollen. He was treated with digitalis tablets with improvement; but later cardiac failure became more pronounced, and he was admitted to hospital one month before the meeting. At that time he was orthopnœic and the cervical veins were distended. The apex beat was in the sixth left intercostal space, 15·0 centimetres (six inches) from the middle line, and a systolic thrill was palpable in this region. Auricular fibrillation was present, the ventricular rate being 96 per minute. A blowing systolic murmur was heard best at the apex, where a diastolic murmur was audible with difficulty. The second sound at the pulmonary area was accentuated. An aortic diastolic murmur was not present. The edge of the liver was palpable three fingers' breadth below the right costal margin, and pitting œdema of the sacrum and legs was present. The electrocardiographic examination revealed auricular fibrillation and right ventricular preponderance. X ray examination in the anterior position disclosed that the enlarged left auricle was visible on the right border of the heart shadow and that there was filling in of the pulmonary arc. Hilar con-

gestion was pronounced. In the right oblique position, with barium in the œsophagus, the enlarged left auricle was outlined and the aortic and pulmonary artery impressions were seen. The right ventricle was enlarged and the prominence of the conus was easily visible.

Tincture of digitalis, in doses of 1·2 cubic centimetres (twenty minims) had been given three times a day since his admission to hospital, so that the ventricular rate had fallen to 76 per minute and the mitral diastolic bruit could be heard more easily. "Salyrgan" had produced a pronounced diuresis and the œdema had disappeared, although the liver was still enlarged. Breathlessness had diminished and the patient felt considerably better.

Pulmonary Abscess.

Dr. Rothstadt, on behalf of Dr. L. E. Hurley, next showed a male patient, aged thirty years, who had been admitted to hospital on March 16, 1938. Seven days before his admission he was talking at dinner when he felt a piece of meat slip "down the wrong way". He became very breathless and gasped until he felt the meat pass on. A few hours later he had pain behind the sternum; this increased in severity, and next day he was unable to swallow because of pain. During the next three days, however, he was able to swallow soft food. Three days before his admission to hospital the pain spread over the front of the chest; he had a cough with mucoid sputum, and he was shivering. On admission to hospital his temperature was 39·2° C. (102·6° F.); but physical examination disclosed no abnormality. X ray examination revealed very slightly diminished translucency of the right upper lobe. During the next few days the patient coughed up blood-stained sputum in increasing quantities.

On March 21, 1938, the percussion note was dull over the right apex posteriorly, where the breath sounds were tubular and the vocal resonance was increased. X ray examination confirmed the presence of consolidation of the posterior portion of the right upper lobe. Bronchoscopy was performed, and thin pus was aspirated from the right upper lobe bronchus; but no foreign body was found. Culture of the sputum revealed the presence of mixed cocci. The physical signs persisted for the next ten days, when X ray examination revealed that the lower half of the right upper lobe was clearer, but that there was a dense shadow of consolidation at the apex. Immediately behind the clavicle there was a translucency, apparently air in a partly filled cavity. The temperature was still elevated, but was not above 37·8° C. (100° F.). The sputum was offensive and the patient was coughing up as much as 400 cubic centimetres (fourteen ounces) each day. He was lying on the left side; the left shoulder was raised by a pillow, and the right shoulder was forward. A mixture of potassium iodide and creosote was administered. X ray examination on April 11, 1938, revealed consolidation at the apex of the right lung; and there were small translucent areas, but no definite abscess cavity. Postural drainage was continued. The sputum diminished to about 60 cubic centimetres (two ounces) per day and was not offensive. On May 2, 1938, further X ray examination revealed diminution in the area of consolidation; and no definite abscess cavity was visible. Bronchoscopy was again performed and lipiodol was injected, but a cavity could not be demonstrated. At the time of the meeting there were about 30 cubic centimetres (one ounce) of sputum each day. The temperature had settled down, and the only signs were relative dulness on percussion and bronchial breathing at the apex of the right lung.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

McMahon, Patrick Joseph, M.B., B.S., 1934 (Univ. Sydney), Auburn Street, Moree.

Dryer, Albert Thomas Benedict, M.B., B.S., 1938 (Univ. Sydney), Parramatta District Hospital, Parramatta.

Proceedings of the Royal Commission Appointed to Inquire into Matters Pertaining to National Health Insurance.

THE Royal Commission on National Health Insurance resumed sitting on Monday, August 29, 1938. Mr. L. S. Abrahams, K.C., opened the case on behalf of the British Medical Association.

Mr. Abrahams: Our grounds of opposition to the rates suggested by the Government and by the Insurance Commission fall into four main headings:

1. That the rate will necessarily lower the quality of service and reduce efficiency.
2. That it will destroy the status of the general practitioner as a professional man doing work of the utmost importance and of great responsibility.
3. That it will lower the emoluments that he is fairly entitled to receive for his work.
4. It will tend to prevent him from rendering the beneficial services he now renders to the community in the form of honorary hospital and charitable work.

It is submitted that quality of service is the very essence of the scheme of national health insurance, and that is recognized by the Government. The Treasurer, Mr. Casey, in the course of his second reading speech on the Bill, which is reported in *Hansard* of Friday, May 6, at pages 803 and 804, said [reads from *Hansard*]. Our case will be that the family doctor is the foundation of the whole medical structure. We are dependent on him for the number of people admitted into hospitals, the work done there, the number retained in the hospitals, and the cost of administration; and that quite apart from the work he does in the prevention of suffering to his patients. The solvency of the sickness benefit fund will depend on him because by proper medical attention he will prevent unnecessary calls on the fund, and by virtue of his skill he will not give unnecessary certificates which would cause the fund to be depleted. Our submission is that the amount the doctor is paid will tend to determine the time which he will be able to give to his work. I propose to tender evidence to establish the claim that if a general practitioner spends time in giving efficient service he will not be able to make a reasonable living according to Australian standards. Evidence will be given as to the number of services that can be given in, say, an hour, having regard to the voluntary hospital work and to the research work which medical practitioners may be expected to undertake. We take it to be an axiom that doctors are still to continue their research work, and to attend to hospitals in a voluntary capacity. Therefore, in estimating the number of services which a doctor can render in a given time, we assume that he is still to take part in these other activities. It may well be that if a doctor's consulting room were full in an evening he might be able to see six people in an hour and attend them efficiently, but there will be always a residuum of work in respect of the patients that he has seen that evening, such as telephone calls in connexion with prescriptions to be sent *et cetera*,

besides possibly work associated with the getting of patients into hospital. Then there are those matters which arise from the carrying on of a practice. The doctor may have to order a new tire for his motor car, or drugs from a wholesale house. All matters connected with the carrying on of a practice in Australia must be taken into consideration, though, of course, the most important things are research work and hospital work which doctors are now doing. Many medical practitioners will be called before the Commission to give evidence, and some of them will give evidence regarding research work upon which they are engaged. In estimating the number of services which a doctor can render in a day of eight hours one must take into consideration all those activities which he has been in the habit of carrying on in the past because, as Mr. Casey said, the very essence of the scheme is that those activities and services which characterized the work of the medical profession in the past shall continue in the future.

We further contend that the quality of service rendered under national insurance in England is not comparable with that given under friendly societies' schemes in Australia. In England the tendency has been for the panel doctor to regard himself as a mere clearing station for hospitals. In the case for the Government, four main matters were put forward. One suggestion was that the amount budgeted for by the Government should be given some weight. We submit respectfully that that is not so. If a proper rate is, say, £1, and 11s. has been budgeted for, then this Royal Commission, if it is to take into consideration the amount budgeted for, must find 11s., in which case the sittings of the Commission will be futile. There can be no half-way course. If the point made regarding the amount budgeted for cannot be given its full weight it cannot be given any weight. There can be no splitting of the difference between 11s. and £1, for instance, and arriving at some such figure as 15s. 6d. on that basis. The terms of reference require the Commission to determine a proper rate irrespective of what has been budgeted for. Otherwise the Commission would have to find that what has been budgeted for is the proper amount.

Another point that has been put forward, tentatively at least, is that some agreement was made by representatives of the medical profession in conference with representatives of the Government. Evidence will be given if necessary that no agreement was made. We gather from the evidence of Mr. Brigden that he conceives that no binding agreement was made, and it appears from the letter signed by Dr. John Hunter that that is the situation. However, we propose to adduce evidence to show that the Government was apparently not seized of the full facts to lay before representatives of the profession. We propose to show that the figures on which the calculations were based are entirely inaccurate. The figure of 3·2 put forward on behalf of the Government to members of the profession is, we will show, absolutely unreliable. The actual figure is nearer 3·5. There are other matters which were not present to the minds of the representatives of the medical profession and the representatives of the Government distinguishing the position based on a pure mathematical calculation from the actual position.

That brings me to the third point, namely, the calculation of the capitation rate fixed by reference to contract practice. I informed the Commission that we proposed to attack the mathematics of that calculation. We attack the figure of 3·2 and the constituents of that figure of 3·2, that is, the figures used in arriving at it. We do not accept those figures. A census has been taken which, we have been informed by actuaries, is entirely reliable, and this census supports our claim. According to the census which covers 10,000 friendly societies in Victoria, we are justified in challenging the figures submitted by the Government. By the time the Royal Commission hears evidence in Victoria we shall be able to show accounts for all the members covered by the census, and this evidence will be supported by statutory declarations in every case. This census shows that the proper figure should be 3·5, or it may be slightly more. We will tender evidence from various practitioners showing their own figures taken from inquiries in every case from members of friendly societies whom they attend which also support the figures arrived at by the census. That is a matter of pure mathematics, but there is a much wider and stronger ground for attacking the calculations based on contract practice. We attack that method because it loses sight of a main relevant fact, namely, that contract service is a concessional service rendered to poor, thrifty and healthy members of the community. If 3·5 is the correct figure, and 600,000 is the correct number of members, there are 1,500,000 persons at present attended to under contract. Doctors will have to attend 1,450,000 new persons under the insurance scheme. It may be assumed that the dependants of the 600,000 people who will now be released for private practice will be useless from the point of view of income for private medical practitioners. They are poor people who have been in the habit of being attended to under contract. It is not to be expected that any considerable body of those dependants will be available for the field of private practice at more than contract rates or more than the national insurance rate. The capitation rate of 11s. works out at 2s. 9d. each on the 3·2 figure taken in England for each service. As I have said, it is not to be expected that the introduction of national health insurance will set free the dependants of those persons at present attended to under friendly society practice for private practice at a rate higher than the contract service rate or the national insurance rate, and that apart from the fact that the Government is proposing to introduce a voluntary scheme to cover the dependants of insured persons. The position therefore is that the doctors will have to attend to 1,500,000 new persons, so that nearly 3,000,000 persons will be out of the field of private practice except for excluded services. Of those over 2,000,000 will be earners, representing over 70% of the bread-winning population. At the present time doctors attend to only a little more than 20% of the bread-winning population under contract service. The importance of this is that the field of private practice will be materially reduced, and this prevents the application of a mathematical formula based on a concessional service to a small proportion of the population. At the present time the doctors who give this concessional service have left to them a large field of private practice, but if this field of private practice is to be taken away by the bulk of the community coming under a fixed rate of service the remuneration of the doctors will be greatly reduced.

The next point put forward on behalf of the Government and Insurance Commission was the rate fixed by reference to the sum of 11s., which is the rate in England. If that is taken as a guide, the fee here should be 30s. because of matters to be taken into account in comparing English with Australian conditions. There is the difference between the standard and cost of living and expenses of medical men in the two countries. That may add an amount which is similar to the exchange rate, namely 25%. There is, however, the difference in the status of the medical man in Australia and the medical man in England. Figures referring to the standard of living will be adduced, as well as figures relating to the income and expenditure of a medical man in Australia. On the subject of status it will be shown that a panel doctor in England is in effect a mere clearing station for hospitals. In hardly any case is a panel doctor on the honorary staff of a hospital, while in 50% of cases medical practitioners in Australia are on the honorary staff of hospitals. In fact, the position of the panel doctor in England is such that he is almost a mere hack. Another matter which distinguishes the panel doctor in Australia from the panel doctor in England is that the responsibility of the doctor in Australia is much greater because of his geographical situation. In the country here large distances have to be travelled by doctor and patient, and the doctor must make up his mind without skilled assistance. There are no specialists outside capital cities, while in England there are specialists in even quite small places. One English panel doctor who practised in Plymouth will be called, and he will show that there is a large number of specialists in that town. Moreover, Australian hospital provision is not as adequate as is hospital provision in England. Evidence will be given that in England when a case shows the slightest sign of difficulty or complication, the patient is sent to a hospital, where he is received. In Australia hospital accommodation is relatively smaller,

and doctors will say that, except in cases which may be regarded as more or less necessary for hospital treatment, it is impossible to get a patient into hospital.

In Australia there is no wide gap between the general practitioner and the specialist in knowledge or in skill; but the position is different in England. The report of the Royal Commission on National Health Insurance in England dated 1926, at pages 130 and 131, has this to say on the subject [reads from report]. That indicates the wide gap between the panel doctor and the specialist in England, but that does not exist here because of the responsibility which must be taken in Australia by the general practitioner.

Another difference is in respect of the quantum of service. Hospital provision in Australia is not so ample and therefore the panel doctor, particularly in the country, will have to render a much greater quantum of service for his capitation fee than is rendered by the doctor in England. Another point we are told by panel doctors is the difference in the temperament and requirement of patients in the two countries. Anyone familiar with the A.I.F. would perhaps be aware of that. In England, before national insurance, the average patient was satisfied with a low standard of service. I say that advisedly, and the statement is supported by the report of the Royal Commission on Health Insurance, which at page 188 states [reads from report]. One can imagine that the procedure adopted by some panel doctors in England would not be tolerated in Australia. For instance, it is the custom with some panel doctors in England that, when time is up, even though the surgery may be full, he will refuse to see any more patients. That could not happen here.

Evidence will be called to show that the private earnings of doctors in England are much lower than those of doctors in Australia. One panel doctor will give evidence to this effect. He was a locum at Manor Park, which is in the East End of London. He was also a locum in a panel practice at Cambridge, which is a good class district, and after that he was for five years in practice for himself in Plymouth, an industrial area.

That five years expired two years ago; so that experience is comparatively recent. We shall show that the fees received for private practice by a doctor at Manor Park are as follows: for a visit or consultation at the doctor's surgery, including a bottle of medicine, 1s. 6d.; for a visit out, including a bottle of medicine, 2s. 6d.; at Cambridge, which is a high-class area, while he was locum, the fee received by this doctor for a consultation in his surgery, including a bottle of medicine, was 2s. 6d.; and for a visit out 5s., plus the bottle of medicine, which had to be delivered by the doctor, and was delivered, in fact, by the doctor. In the latter case he charged 5s. a visit. That is, he went out, saw his patient, came back in the middle of the day, made up his medicine, and sent out the bottle of medicine by a boy. In Plymouth the fee received was 2s. 6d. for a consultation at the surgery, and 3s. 6d. for a visit. The result was that with 1,800 people on his panel he earned about £800 a year from his panel practice. Dividing that 9s. by the figure we have been given, namely, 3·76 (that is, we have been told there are 3·76 services for each person on the panel), the resultant figure is 2s. 5d. per service, which corresponds substantially to his average from a private patient, when you allow for the fact that he supplies medicine. So that his 9s. gives substantially an equivalent fee to what he gets from private practice. If we are to get four visits, which we suggest is a proper figure, per service, based on something like we are getting now, and multiply by four as in England, to fix the capitation rate, I suggest the capitation rate will come out to something like 30s. Evidence will be given in this respect by doctors who have had panel experience.

The Chairman: If the customary fee in Australia, whatever it may be, is to be taken as approximately the measure, then it would appear that in lodge practice medical men have been giving their services for less than half.

Mr. Abrahams: That is so.

The Chairman: Well then, one has to recognize that in the case of the professions as well as in the case of other work, the price ultimately is fixed by the demand, and the demand of the persons requiring their service and their capacity to pay. Presumptively lodge practice, by and large, has been paid for at the price fixed by market conditions.

Mr. Abrahams: The view taken by the medical profession, and evidence will be forthcoming in support of it, is that that is a concessional service fixed with reference to a class of people who cannot afford to pay. We are quite satisfied to stand or fall by a market demand based on what we have been paid by the people, now coming under national health insurance, in the past. If that fixes the market price, then we can give this Commission the rate actually paid for private practice by the people now coming under national health insurance. A contract practice is a service rendered to people who would otherwise drift away to the hospitals. There can be no question about this. Everyone realizes that it is a service rendered to the poorer sections of the community, and it will be put that it is a concessional service, but the fees which we shall show to the Commission will be the fees actually paid per service in the past by the very people coming under national health insurance.

The Chairman: You may surprise me as to the willingness, or the motives, of medical men in doing lodge practice; but I must confess I have never known the average practitioner to be averse to lodge practice; he has not turned it away, or shown any disinclination to take it, and I have always assumed that he regards it not only as a means of furnishing valuable service to the community, but also as a means of getting something like a solid foundation for his own income.

Mr. Abrahams: There can be no doubt about that. The matter has to be looked at in this light: it has been stated that in England certain honorary work is rendered for nothing, the work rendered for the hospitals; it has to be conceded that that is not rendered solely out of a sense of public duty. No one is suggesting that honorary work in England is valuable to young doctors and old doctors, as is the case here, in letting the quality of their work be seen by students and others. While in Australia it is felt there is a public duty involved, because we see the doctors remaining on after all necessity for their service disappears, while such service is done partly out of a sense of public duty; nevertheless in the early stages of a doctor's career such service has its distinct advantages, and the same thing may be said of lodge practice. In the early stages it forms the nucleus to the young struggling doctor, but afterwards, I venture to suggest respectfully, that the most successful doctors, apart from their sense of obligation, would be very happy to be rid of it. It has to be realized that both aspects enter the matter, but the main point I wish to stress is that it relates to a small part of the community.

May I put this illustration. In New South Wales we have a system of legal aid under which those barristers who take part in it are required to do the work without fees. Many barristers are happy to perform that service, but if they were required to do it in respect of the seven-tenths of the earning community, I think something would be said about it.

Mr. Dovey: They are not as anxious to do it as the doctors are to get honorary positions in public hospitals.

Mr. Abrahams: Maybe not; but no one would suggest that those doctors who have become famous in their profession in Australia and who continue to do this work, so continue to do it because they hope to gain some monetary advantage.

The Chairman: Of course not.

Mr. Abrahams: The real test of this practice is what proportion, relatively, does it bear to the whole field of practice, and it is in that way we propose to tackle the problem and to call our evidence. We can well afford to do the private practice at 9s. per service, but if that figure is to be fixed in respect to a large proportion of

the community, then the standard of service and the emoluments of the doctors will be lowered. My object in dividing the 9s. by 3·75 is to show a comparison in England between private earnings and panel earnings, and also to draw a similar comparison between private earnings and the proposed panel earnings in this country. We intend to submit actual evidence both to attack the figure of 3·2, and any other matter which we do not accept, and also to put forward figures supporting the loss of private practice in order to show what the proposed capitation rate should be in order to ensure reasonable remuneration. We had hoped to base this actuarial evidence solely on the result of the *questionnaires*, but they were not drafted very happily and led to considerable confusion. In view of the remarks made earlier in the proceedings, we propose to call individual doctors to give evidence on oath and to base the actuarial figures, at any rate in part, on the result of their evidence. They will have to be asked to give evidence, among other things, pertaining to the cost of equipping and acquiring their practice, their income and expenditure, for the purpose of founding the actuarial figures. The doctors we propose to call are representative of all types of practice in each State. We propose to call them from city, suburb and country, and so far as we have been able to judge, as representative of every type of practice. They are not selected with the object of putting before the Commission only the poorer class of practice. If anybody, including my friend, is of opinion that any doctor called does not represent a fair sample, and suggests that he can call somebody from anywhere else, so far as we are able to do so, we shall comply with such a request. The situation in this country is that the population in the main consists of working men and their families, and when one looks around Sydney one finds that there are very few practices which do not consist in the main of patients who are either working people themselves or the dependants of working people. Wherever you go in Sydney you will find that that is the situation.

However, we have interviewed witnesses from a suburb like Vaucluse, which is a very high-class residential suburb in Sydney, and a witness from that suburb will be called. We have got another from Mosman, which is also a high-class residential suburb, who will tell this Commission that his patients consist mostly of older people who are retired and will not come under the scheme.

Mr. Dovey: What I understood before the adjournment was that my friend was going to have a number of *questionnaires* sent out to every medical man in New South Wales. No names would appear on the *questionnaires*, but there would be a key to the names and we would be supplied with the key. My friend suggests that he proposes to call as witnesses, selected more or less at random, representatives of particular kinds of practice. I should think it would be desirable, and I say this not in any way disparaging my friend or his witnesses, that we should have an opportunity of seeing the names of these gentlemen whom he proposes to call, in order that, should we desire, we could then call witnesses from the list of those who have made answers to the *questionnaires* but who are not called by Mr. Abrahams. I understood that the answers to these *questionnaires* were to be made available to this Commission in some tabulated form at some convenient time.

The Chairman: I anticipated something of this kind, but apparently there seem to be difficulties preventing any complete tabulation of that kind. If Mr. Abrahams produces his samples of individual practitioners and attempts to base an actuarial calculation upon their evidence, I think you should be entitled to call your samples if you have any available.

Mr. Dovey: Our samples will be those picked out of the hat, so to speak.

The Chairman: It is just as well to recognize at once that any actuarial calculation based upon a comparatively few specimens—and they must be comparatively few, even though they are representative—is subject to comment, to say the least of it. It may be said that the field is not wide enough to make an actuarial calculation valid.

Mr. Dovey: It was then decided to put before the Commission a representative body of doctors; when we had had an opportunity to look at the *questionnaires* we were to pick out more or less at random a number of doctors whom we would ask to come along.

The argument as to the method of calling medical witnesses continued for a short period, then Mr. Abrahams spoke as follows:

If my friend wants a list of the names of those who returned *questionnaires* throughout Australia he can have it, and so far as we are able to do so we will call any doctor whose name appears on that list. We have nothing to keep back. We are attempting to arrive at a proper result. My clients do not desire to produce figures which will suggest a result other than the correct result. If my friend can suggest any other method to achieve our aim we will be happy to consider it.

The way in which we propose to estimate this loss of private practice—and it will be supported with actuarial approval—is this: we propose to call representatives of every type of practice throughout Australia. If there are any special types of practice, as on the coalfields in New South Wales or on the goldfields in Western Australia, evidence of those will be brought before the Commission. They are in a special category so far as we can ascertain. In respect of the coalfields in New South Wales, it is all contract practice and the loss is much more easily ascertained in those cases, and, of course, is much heavier in proportion to the volume of earnings. The first matter to be taken into consideration in attempting to get at this loss is the method which can be adopted, and, finally, we have come to the conclusion that a loss can be put in one of two ways: either you have to work harder for the same money, or you have to do the same work for less money. The way we propose to put it before the Commission is a method based on exactly the same amount of work for each doctor. In that way we will arrive at a figure which shows a loss under the national health insurance on a capitation rate of 11s., based on the same amount of work. The Royal Commission will then be able to say, firstly, what the rate should be for the same work; or, secondly, how much extra work is involved, and therefore how many panel patients a man will have to have in order to earn the same money he has earned previously. In either view it is desirable in the first instance to put before the Commission the figures based on the loss which will be suffered assuming the same amount of work is done. For that purpose we have found—and evidence will be given to this effect—that it is only gross income which matters. The expenses are more or less constant. It is perfectly true that if a man has more visits in a particular year he will use his motor car a little more, but those differences cannot possibly affect the result when we come to deal with the whole of Australia. It will be shown that minor matters of that sort do not affect the matter. We propose to take a particular year, namely, that for which the doctors have complete figures, and it will be found mostly to be the year ended June 30, 1937; although for some individual places that year cannot be taken, for example, where a doctor has had an illness which has kept him off work for a couple of months, in which cases another year is selected. In other instances they have not got complete records for the particular year. For instance, it has been found that the number of services given to lodge patients in some instances has never been kept until the doctors were asked to keep it, as from January 1 this year. So if they had to take the year ended June 30, 1937, they have been told so far as lodge services are concerned to take the best six months of this year if that period is typical of their lodge service during previous years, and to multiply that figure by two.

We propose to take the gross income for the year selected and split it up into the capitation fee received from lodges and all other remuneration to ascertain the number of friendly society members and the services rendered during the year. We thereby get the rate per service. In regard to other income, which includes services to lodge patients, we take the total number of services rendered for the balance of the income and find the rate

per service. We then ask him to estimate the number of private services fewer which will be rendered under national health insurance. That can be fairly reliably calculated. Supposing we had a figure of 8,000 services, 2,000 of which are lodge patients and 6,000 private patients. If a man says the diminution of his private services will amount to 33⅓%, it will be seen that he will lose 2,000 private services. We know that he will lose two-thirds of his lodge services because we know that two-thirds of the friendly society patients will come under national health insurance. We know the number of services he will render under national health insurance and if we subtract that from the number he now renders we will have the number of services to panel patients. Dividing that by four will give the number of panel patients he will have and consequently the income he will receive at the reduced rate, or alternatively, the extra services he will have to render to make up his income. I shall produce a form to the Commission to show how the estimate is arrived at. The first thing to be done in order to estimate the loss of service in a particular year is to count heads. We know that the average loss of persons coming under national health insurance, compared with the population, will be about 30%. In the higher class of practice the diminution might be only 5%. In the more industrialized suburbs there has to be something to make up for this low diminution, and we should probably find it rising to about 40%. But a count of heads is not the only method to be considered. It has to be remembered that a doctor will lose patients who come under national health insurance only in respect of the included service; he will still have them in respect of excluded services. This count of heads is only the first figure. We know the average for the Commonwealth. We have asked the doctors to estimate on a count of heads what percentage they will lose. Supposing he arrives at the figure 30%, counting heads, he still has to say "I have these persons in respect of matters that come within the excluded services, such as anæsthetics, operations, workers' compensation". Confinements we regard as negligible because few married women will come under the scheme and it is thought that those who do will not be having any children. But there is a wide field in workers' compensation, operations, and perhaps serious fractures. Some diminution of that 30% must be allowed for in respect of that work. Most of them have arrived at that by taking over a particular period the total number of services rendered. It is very often found to be in the vicinity of 20%, although it varies in different practices. Some doctors may have a bigger operative practice and some may specialize in a particular type of work, but generally it may fairly be supposed that about 20% of a general practitioner's services are rendered in respect of the excluded matters. Then there are other considerations which operate in the other way. The tendency will be to leave to private practice the dependants of national health insurance patients. Miners in the Newcastle area who have a set rate fixed for them will not pay for their dependants. There are set rates on the Newcastle coalfields in private practice and they do not include confinements or children over sixteen years of age; but the miners refuse to pay for a child over sixteen years and in many cases for a confinement. Out of sixty cases of confinements there was a refusal to pay in thirty. That, of course, is a special area. But one wonders how far there will be a tendency for the dependants of people who come under national health insurance not to go to the panel doctor. If a man has to pay he may prefer to go to a specialist or to a hospital. These matters have to be taken into consideration in re-raising the percentage. It has been thought better in these matters of speculation to be on the conservative side. The reduction is more or less mathematical, but we suggest that the Commission will have to take these matters into consideration. It is proposed to ask every witness to fill in a document so that the Commission may have it as an exhibit. It may be said that in the year ended June 30, 1937, payment was not made for all the services rendered in that year; there is a brought forward from the previous year and a carry forward into the following year. The evidence will indicate, however, that in nearly every case a doctor's practice does not fluctuate very much. It is thought that the carry forward will equal the brought forward and that it will be fair to take the receipts and the services rendered in a year. Where the number of dependants is known the figure will be inserted; in many cases it is not known. Assuming the Government's figures to be accurate, we submit that the figure two-thirds should be taken in the case of every practitioner. In some districts there will be more panel patients than can be comfortably handled, whereas in country districts there will not be enough. A doctor may have patients who live twenty miles away and prefer to be treated by him, but the nearest town may be only a few miles away and under national health insurance they will be obliged to go there. We found very many problems in regard to distribution, but under the scheme as we have framed it they will be eliminated. We know what services are being rendered and will have to be rendered or, if the same services be rendered, the diminution of income. The difference between a mere mathematical calculation and the true position is that caused by the swing over from the field of private practice of a large preponderance of wage earners. Our calculation may not be mathematically accurate to 1%, 2% or 5%, but it will make very little difference even if the estimate of diminution were mistakenly calculated to as high as 5%. By this system error is reduced as far as it can be. We know that it is not perfect, but we submit respectfully that it is as nearly accurate as it can be in the circumstances. It will be found that for the most part the wives of friendly society members also are examined by the same doctor. In many cases the income limit clause of the agreement is enforced by the doctor. I understand also that lodge secretaries prevent some men from going to doctors because they know that they will not pass. Many practitioners charge a night fee as a deterrent against night calls. In one case that I can think of a doctor had many night calls until he enforced the night fee, when the calls ceased.

There are some matters upon which I desire to obtain Your Honour's guidance. One matter relates to excluded services. The report of the Commission will be based on the view that a regulation will later be passed excluding those services which we have been told are to be excluded. It has been suggested to me that in all probability the Insurance Commission has no power to exclude cases falling under workers' compensation. Where will the doctors be if this Royal Commission proceeds on the basis that workers' compensation is still to be left in the field of private practice and it is found that by law it cannot be left in that field? In my opinion great difficulty arises in regard to the interpretation of Section 47 of the Act. If you exclude a thing as a medical service it is most difficult to define it according to the person who is compelled to pay for it. A confinement is mentioned as a medical service. The question is, can you say that there are three sorts of medical services? It is most difficult to say that a fracture is in one case a medical service—that is, where it arises not out of a person's employment—but if it arises in the course of his employment the attendance ceases to be a medical service.

After some further discussion Mr. Abrahams said that he was prepared to call witnesses.

The Chairman: Do you want this evidence taken in camera?

Mr. Abrahams: Yes.

Mr. Dovey: A great many statements have been made by my friend which will go out to the public, and if the evidence of the witnesses which he proposes to call is to be heard in camera there will be no opportunity to break down the effect of such evidence or of the statements he has made. Many people and organizations are interested in this subject, and the only way in which they can be encouraged to come forward and give evidence is to have the proceedings of the Commission brought to their knowledge. I agree that it is undesirable for a doctor to give evidence regarding his own income other than *in camera*, but evidence regarding all other matters should

be given in open court. I submit that evidence should be given *in camera* only regarding income from the practice which a doctor is conducting at the present time.

The Chairman: I agree that in respect to many of the matters touched upon by Mr. Abrahams in his opening there is no need for evidence to be heard *in camera*. Perhaps the best course would be to proceed openly until the necessity arises for hearing *in camera*.

Mr. Abrahams: If any question is asked in cross-examination which appears to touch on the income of the witness or of any other doctor I shall object unless the evidence is heard *in camera*.

Mr. Dovey: That is understood.

Dr. Norman Ernest Davis, of Manly, was sworn and was examined by Mr. Abrahams in open court and later *in camera*.

The Commission adjourned until 10.30 a.m. on Tuesday, August 30, 1938.

Tuesday, August 30, 1938.

The Commission resumed sitting on August 30, 1938. The examination of Dr. Davis was concluded, and evidence was obtained from Dr. Thomas Henry Small.

STATEMENT PREPARED BY THE NATIONAL HEALTH COMMISSIONERS.

MR. J. B. BRIGDEN tendered as evidence before the Royal Commission a statement prepared by the National Health Commissioners. Parts of this statement appeared in the issues of August 20 and August 27, 1938.

The Council did not admit that any allowance should be made for the fact that the insurance medical service would be free from control by Friendly Societies and claimed that they had that freedom under the existing lodge practice. The only improvement in the insurance service would be prompt payment and freedom from bad debts.

After further discussion, the Council suggested that there might be some advantage in excluding from the scheme the administration of general anæsthetics. The doctors would not be asked to accept less than the well-established minimum fee of £1 1s., and the patients would not feel that they were being given a cheap service. They agreed that it would be important to check the excessive use of anæsthetics, to attract additional fees, and undertook to supervise claims upon insured persons for this service.

At this stage the Commonwealth Treasurer joined the discussion. The offer made by the Federal Council was reported: 9s. for the lodge practice range or 12s. for a wider service, together with the Council's view that the mileage limit should be two miles instead of three and that the position should be reviewed after three years.

The Treasurer said that the money available would not permit of a payment of more than 11s. capitation fee, with an additional 1s. a head (roughly £100,000) for mileage, and pressed the desirability of providing a wider service and that any agreement reached should remain in force for five years.

The representatives of the Council said that they could not accept 11s. for a wider service, but asked time for further consideration. The Council representatives present had plenary powers and could reach an effective decision.

On resumption of the discussion, the representatives of the Council said that they were disposed to accept the offer for mileage, though they thought that that matter should be reviewed at a fairly early date ("five years or earlier"). They would also accept 11s. for a service including fractures, dislocations and certain minor operations and for anæsthetics they would agree that a fixed rate of £1 1s. should be prescribed and that a doctor proposing to charge that fee should notify the appropriate Deputy Commissioner.

The Treasurer thought that the figures did not justify 11s. on this basis and suggested as an alternative 10s. 6d. without anæsthetics, the remaining 6d. to form a pool from which accounts for anæsthetics should be paid. After discussion, the representatives of the Council said that they were unable to accept this suggestion, and that their minimum figures were 11s. without anæsthetics.

Further adjournments and discussions followed, including discussion as to the exact range of anæsthetic that was at issue—all ordinary inhalation anæsthesia would be extra, as would be spinal anæsthesia. The maximum extra fee of £1 1s. was agreed provisionally to cover all excluded anæsthesia except where required for a hospital operation. Excluded anæsthesia would be that for which the services of a second practitioner were required.

The Treasurer accepted the offer of the service, excluding anæsthetics, for 11s., with the prescription of a flat rate for anæsthetics as explained and the profession's help in controlling excessive charges for anæsthetics.

[*Copy.*]

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney, New South Wales,
2nd April, 1938.

(JGH/CSN).

Sir Walter Kinnear,
National Insurance Department of the Treasury,
Commonwealth of Australia,
Canberra, F.C.T.

SIR,

National Health Insurance.

I am directed to advise you that the decisions made at the conference held in Melbourne 10th and 11th March, 1938, and Sydney 28th and 29th March, 1938, between representatives of the Commonwealth Government and the Executive Committee of the Federal Council of the British Medical Association in Australia in regard to National Health Insurance were as follows:—

(i) (*a*) That the capitation fee for a general practitioner service, excluding treatment in respect of a confinement and treatment of an insured person who becomes entitled to the benefits of any Commonwealth or State Workers' Compensation Act or Seamen's Compensation Act, and excluding major operations and anæsthetics, be eleven shillings.

(*b*) That the rate of payment for administering an anæsthetic to an insured person, unless the administration is in itself a specialist service, be one guinea.

(*c*) That where it is required to administer an anæsthetic to an insured person the insurance practitioner on whose list the insured person is placed shall within 10 days notify the District Medical Benefit Committee of the fact.

(*d*) Anæsthetic shall be deemed to mean inhalation anæsthetic, and the fee for the administration of such anæsthetic shall, excepting where the circumstances are such that it is not reasonably possible to obtain the services of another practitioner, be payable only where an anæsthetic is administered by one practitioner and the treatment afforded by another practitioner.

(ii) That mileage be payable at the rate of two shillings a mile one way beyond three miles but that in areas presenting special travelling difficulties the rate be increased, and that the question be reviewed in five years.

With regard to anæsthetics it has been found difficult to define what constitutes an anæsthetic for the purpose of the Scheme. The definition submitted, although somewhat cumbersome, appears, however, having regard to all the circumstances, to meet the case.

The Association through its Insurance Acts Committees will do all in its power to see that there is no unnecessary administration of anæsthetics.

However, once again the Council would emphasize the many advantages of a complete service, including anæsthetics.

Faithfully yours,

(Sgd.) J. G. HUNTER,
General Secretary.

EXTRACT FROM THE MEDICAL JOURNAL OF AUSTRALIA (VOL. I., NO. 16, PAGE 719) OF THE 16TH APRIL, 1938.

(Here follows a copy of the statement under the title "Medical Benefit under the National Health Insurance Scheme", which was published in the issue of this journal of April 16, 1938, at page 718.)

EXTRACT FROM THE MEDICAL JOURNAL OF AUSTRALIA (VOL. I., NO. 20, PAGE 859) OF THE 14TH MAY, 1938.

(Here follows a copy of the leading article that appeared in this journal in the issue of May 14, 1938.)

[*Copy.*]

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.[1]

135 Macquarie Street,
Sydney, New South Wales,
21st May, 1938.

(No. 1228).

The Honourable R. G. Casey, D.S.O., M.H.R.
Parliament House,
Canberra.

SIR,

National Health Insurance.

Since the conference between the representatives of the Commonwealth Government and the Executive of the Federal Council of the British Medical Association on the medical benefit section of the National Health and Pensions Insurance Bill concluded, the agreement reached has been placed before the medical profession in Australia who are members of the British Medical Association, and an opportunity has been afforded them of discussing the Bill.

You will remember that the Executive of the Federal Council, when discussing this matter with the representatives of the Commonwealth Government, distinctly stated that it was not in a position to bind the profession to accept the terms of the agreement. It was influenced in this respect by the fact that the details of the Bill were completely unknown at that time to the profession as a whole. It was felt that it was only reasonable that the members should be able to place their views before their representatives and give the benefit of their knowledge of medical practice so essential to a successful working of the scheme.

Whilst no objection has been made to the inclusion of medical benefits in the Bill, there are many features of the proposed terms of service which appear to be unsatisfactory to the profession and which undoubtedly would affect the smooth and efficient working of the service. A brief and categorical statement of these points follows herewith:

1. *Capitation Fee.*—This is regarded as being totally inadequate. The minimum rate that would be acceptable to the profession is 14s. in the Metropolitan Areas with an additional 25 per cent. for Country Areas. In Australia the country practitioners have always been paid at a higher rate under contract practice than the city practitioners, the average difference being between 25 per cent. and 30 per cent. The reasons for this briefly are—

(*a*) the isolation of the country practitioner and lack of amenities;

(*b*) the irregular hours of working of country practitioners;

(*c*) the fact that the country practitioner, unlike the city practitioner, is unable to transfer his difficult cases to a large hospital; and

(*d*) as an encouragement to doctors to go out into the sparsely populated areas. The provision of medical services in these areas is a very real problem throughout Australia;

(*e*) in certain areas the additional cost of living.

2. *Mileage.*—This is also regarded as being inadequate, the minimum rate which would be acceptable being 2s. 6d., 6d. of which should be payable by the patient. Under contract practice in Australia, mileage is always payable by the patient as a deterrent against unnecessary calls. It is also considered that mileage should be paid beyond two miles, a radius recognized by Government Departments such as the Repatriation Department.

3. *Night Calls.*—There is universal dissatisfaction in regard to the absence of any night fees. The fee need not be large, 2s. 6d. would be sufficient, and it is not to be regarded as income-producing, but purely as a restriction in order to prevent an excessive amount of night work. The policy of asking the doctor to make a complaint when he considers a call unnecessary or unreasonable does not appeal to the profession. The night fee would be payable by the patient for calls between 8 p.m. and 8 a.m.

4. *Services to be excluded*—in addition to those specified in the proposed terms—

(*a*) Anæsthetics. As anæsthetics are actually outside the terms of service, the view of the profession is that there should be no interference with their relationships with their patients either in regard to the fee or to the type of anæsthetic to be administered.

(*b*) There is universal disapproval of S.50(1) of the National Health and Pensions Insurance Bill and it is thought that the Section should be amended by the addition of the following words, viz.—"except in the case of acute alcoholism and acute venereal disease."

(*c*) Fractures and Dislocations. The profession particularly desires that the more difficult and complicated fractures and dislocations be excluded and that schedules should be drawn up of such fractures and dislocations.

(*d*) Premature Births, Miscarriage and Abortions in the view of the profession should not be included in the terms of service.

It is obvious that the success of the medical section of this scheme depends on the whole-hearted co-operation of the profession throughout Australia and this co-operation will depend largely on the conditions under which the profession has to work. These conditions are not satisfactory in their minds largely because they depart so far from the existing conditions of contract practice throughout Australia.

Having regard to the views of the profession as set out above, the Federal Executive would respectfully urge that you give serious consideration to the whole question of the terms of service.

As you are no doubt aware, one of the conditions of the agreement with the Commonwealth representatives was that there should be a clause in the Bill excluding from any benefits under the National Health and Pensions Insurance Bill any insured person entitled to the benefits of any State or Commonwealth Workers' Compensation Act or Employees' Liability Act. It is noted, however, that, while Section 69 deals with sickness or disablement benefits payable to an insured person entitled to the benefits of any Act or State Act, no reference whatever is made to the question of medical benefits.

As it is not a matter that the Committee considers can be adequately dealt with by regulation, the Committee respectfully requests that, in accordance with the agreement, a clause be included in the Bill dealing with this particular question.

In regard to Clause 115 of the Bill, the Federal Executive would point out that the amount allocated is totally inadequate to provide for the complete medical service envisaged, and therefore respectfully asks that consideration be given to amending this amount.

[1] The substance of this letter was published in this journal under the heading "National Health Insurance", in the issue of May 28, 1938, at page 943.

When the question of medical benefits was being discussed at the meetings in Melbourne and Sydney, it was agreed that X-ray and pathological examinations should not be included within the scope of the general practitioner service.

As efficient treatment by the general practitioner depends on the early and accurate recognition of the nature of the ailment or injury, the Executive is of the opinion that the provision of X-ray and pathological services to insured persons is a necessity. In order to provide such services, it is suggested that a specialist service pool, quite apart from the Medical Benefit account, be constituted, from which payments could be made to the practitioners rendering the services.

In order to give every assistance, the General Secretary, Dr. J. G. Hunter, will be at your service in Canberra.

Yours faithfully,
J. G. HUNTER,
General Secretary.

[*Copy.*]

FEDERAL COUNCIL OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA.

135 Macquarie Street,
Sydney, New South Wales,
13th June, 1938.

(1251).

The Honorable R. G. Casey, D.S.O., M.H.R.,
Parliament House,
Canberra, Federal Capital Territory.

SIR,

National Health Insurance.

You will remember the circumstances connected with the conferences which have taken place between the Executive of the Federal Council of the British Medical Association in Australia and yourself. The conferences commenced at your invitation on 10th March and extended to 29th March, 1938, and the result of the agreement reached was communicated to Sir Walter Kinnear in a letter dated 2nd April.

You will also remember that we were informed that the National Health and Pensions Insurance Bill must be ready for presentation to Parliament some time early in May, and that you requested us, in order to avoid publicity, to regard the terms of the agreement as confidential until a simultaneous statement was made by yourself and us. The statement was made on 16th April. The reason given to us was that the matter must come before Cabinet before it was published. For these two reasons, firstly, the late date of our negotiations in relation to the preparation of the Bill, and, secondly, the necessity for regarding the negotiations as confidential, the time available for the Executive Committee of the Federal Council to confer with the members of the British Medical Association in Australia was unduly curtailed. Hence we informed you that we did not regard this agreement as binding on the members of the British Medical Association in Australia.

We now know, from the general practitioners themselves, their attitude towards the Bill and the proposed conditions of service in the medical benefit provisions and we can state very definitely that there is a general rejection by the members of the British Medical Association in every Branch in Australia of the terms of the agreement.

We also know that the members of the British Medical Association are willing to co-operate with the Government in the implementation of the Bill and the provision of medical services under it, if the terms offered by the Government are acceptable to them.

The terms contained in our letter to you of 21st May would be acceptable to our members.

We desire, however, to inform you very emphatically that, unless such terms or their equivalent are accepted by you we cannot assure you of the co-operation of general practitioners which you and we so earnestly desire. This co-operation is essential to the establishment of a good medical service.

We therefore invite you, in view of the facts set out above, to reconsider the terms of service offered to the medical profession in Australia.

To this end, we would ask that the Prime Minister and yourself receive a deputation at Canberra consisting of the members of the Federal Council of the British Medical Association in Australia on Thursday, 16th June.

We would also request that you withhold consideration in Parliament of all matters concerning the administration of the terms of medical benefit until such time as a workable agreement is reached.

We would respectfully request the favour of a reply at the earliest possible date.

Faithfully yours,
J. G. HUNTER,
General Secretary.

Correspondence.

PUERPERAL SEPTICÆMIA.

SIR: May I be allowed space to thank Dr. P. L. Daniel for the interest expressed in his letter to THE MEDICAL JOURNAL OF AUSTRALIA of June 11, in the paper by Dr. Tewsley and myself, and to make a few comments on some of the points raised by him.

Dr. Daniel asks whether transfer of organisms to the genital tract of parturient or puerperal women from such sources as their own or their contacts' respiratory tracts is proven or only assumed. It would be extemely difficult to demonstrate (even with the aid of opportune cinematography) the actual transit of streptococci; but it does not seem to us an unwarranted assumption to state, after study of the evidence provided by the careful work of the authors referred to in our paper, that such transfer is the "most probable" cause of puerperal infection with hæmolytic streptococci. Therefore we hold that, until there is convincing proof to the contrary, it is reasonable that midwifery attendants should, in fairness to their patients, base their technique on these strong probabilities. In our opinion, measures such as those we have suggested, which include masking, offer in the present state of knowledge the best hope of success in preventing both puerperal infection itself and possible criticism of the attendants.

I agree with Dr. Daniel's contention that we should regard puerperal infection as we would any other streptococcal infection, such as scarlet fever or erysipelas, in so far as it is in keeping with the findings of Griffith and many subsequent workers that streptococci of identical types may be found, not only in the multifarious lesions caused by hæmolytic streptococci, which include scarlet fever, erysipelas and puerperal sepsis, but also in the respiratory tracts of some 7% of the general community, and that cross-infections causing different types of streptococcal disease may occur, often through apparently healthy intermediaries. These findings certainly provide an argument in favour of recognizing the essential relationship of the various manifestations of streptococcal infection, but they also emphasize the need for prevention of transfer of these organisms from person to person, particularly to those, among whom are puerperal women, who appear to be especially susceptible to infection.

With regard to the site of entry of the infecting organisms in puerperal sepsis, in a series of 66 cases tabulated by Dora Colebrook, hæmolytic streptococci were present in the blood stream in four, and in another site (? pleural fluid) in one, without proof that they were also present in the uterus. These findings agree with my own experience that a profuse growth of the infecting organisms can usually be obtained from the genital tract, with or without concurrent growth in the blood or other sites. On the other hand, hæmolytic streptococci are far less frequently present in the nose or throat of such patients (38% in Dora Colebrook's series). Admittedly

these figures do not exclude the possibility that the organisms first gained entrance to the blood stream from the nose or throat, and subsequently invaded the genital tract; but, except perhaps in fulminating cases, bacteriological experience of the tendency of the respiratory tract to harbour hæmolytic streptococci suggests that it is most unlikely that they would disappear so readily as to be unobtainable from this site in 62% of a series such as that of Dr. Colebrook. The most convincing evidence in favour of Dr. Daniel's theory would be the demonstration of pathogenic streptococci in cultures from the nose or throat of a patient (and their absence from all her contacts, social and professional) immediately prior to the onset of puerperal septicæmia, provided that it could also be shown that the bacteriæmia preceded infection of the genital tract. It is obvious that the combination of circumstances in which such data might be obtained must be extremely rare. In the meantime it seems wisest to base action in this respect also on the probability that, according to available evidence, direct implantation of exogenous organisms on damaged tissues in the genital tract is responsible for the majority of cases of puerperal infection with hæmolytic streptococci.

There seems no doubt that patients with puerperal streptococcal septicæmia are a menace to their contacts; but in view of Dora Colebrook's figures, I do not think that the dissemination of organisms from such patients is solely from their respiratory tracts, as Dr. Daniel suggests; nor do I think it is logical, if recognizing this menace, to refuse to recognize the converse, namely, the possibility of transfer of organisms from contacts to patients.

Yours, etc.,

LUCY M. BRYCE.

London,
July 29, 1938.

THE NATIONAL HEALTH INSURANCE EMERGENCY FUND.

SIR: In reference to the letters of Dr. Molesworth (THE MEDICAL JOURNAL OF AUSTRALIA, July 30, 1938) and of "G.P." (THE MEDICAL JOURNAL OF AUSTRALIA, August 13, 1938), I wish to offer these comments.

It is the avowed policy of the British Medical Association in Australia to foster a satisfactory national health insurance scheme, and it is right that this is so. The outcry against the sum of ten guineas asked for is not justified. It is ludicrous to suggest that we, as a body, cannot afford this amount per man to support a fund which has as its object the preservation of our very livelihood.

It may be of interest to Dr. Molesworth to know that in 1912 the British Medical Association in England, when faced with the position that we are now facing, asked its members for twenty pounds. From the results, it would appear that they met with the same response as our finance committee at the present time.

A Royal Commission is sitting and our Association has to be represented. That representation, to be effective, must be supported by the whole body of our profession. The only practical way we can offer our support is to provide the funds necessary to cover the enormous expense which will be incurred.

Yours, etc.,

"ONE WHO HAS CONTRIBUTED."

Sydney,
August 19, 1938.

NATIONAL HEALTH INSURANCE.

SIR: Doubtless it is proper to credit with the best of motives those who desire to change present medical practice for the benefit of our fellow citizens. No less incumbent, however, does it seem to be to study existing changes in practice concurrently with trends which have been revealed over a period of years since the changes were made. Such a study, made by Dr. James L. Halliday, Regional Medical Officer, Department of Health for Scotland, is reported in *The British Medical Journal* of July 2, 1938. His findings are illuminating.

"The most significant feature of sickness among insured persons", he says, "has been its rise over the past 20 years." There is no mistaking the clarity of this statement. There is no escape from its directness. More detailed examination of the figures shows that "in England and Wales the rate of sickness during the years 1921 to 1926 rose by nearly 50 per cent. and that of disablement by nearly 80 per cent." Since 1926 the high level of increase has been maintained.

During this twenty-year period medicine has made notable advances. Signal triumphs have been won over disease-producing causes—physical, chemical and micro-organic. The results include a falling death rate, greater expectation of life, increasing height and weight of school-children, and many other desirable achievements. But, concurrently, there is a very considerable increase in psychogenic illness among the nationally insured.

Halliday uses the term "psychosomatic" to cover both psychogenesis on the one hand, and bodily signs and symptoms on the other. An individual encounters a setting of events which to him means difficulty, strain, upset or frustration. The psychic trauma he experiences leads to alterations in chemistry, rhythm, secretion, even structure—somatic change. Investigations show that a proportion of illnesses with the labels of gastritis, rheumatism, anæmia, debility, peptic ulcer, bronchitis and others implying somatic changes have psychic ætiological factors of fundamental importance.

Psychosomatic illness shows a steady and considerable increase among insured persons. Such is the disturbing disclosure. This leads to a steadily decreasing capacity for work on the part of the insured. May it be considered relevant here to inquire whether there is any cogent reason for anticipating a different result in Australia?

The increase in incapacitating psychosomatic illness is most marked in the younger age-groups. It may be well to correlate this with another trend of today. Recent publications have brought to our notice the fact that in Australia the proportion of young people to old is rapidly diminishing. Caring for the increasing numbers of elder people necessitates more effort on the part of the diminishing numbers of younger folk. As a matter of fact, however, it is among the younger that incapacity is shown by Halliday to be increasing.

Psychosomatic illness, says Halliday, should be dealt with by general practitioners rather than by those practising in psychiatry alone. There is considerable agreement with this opinion among psychiatrists themselves. Indeed "a large part of general practice consists of illness which, though it may seem to have an organic pathological basis, is psychogenically conditioned"; some say a third, some much more. And it does not seem to be decreasing. Your editorial of August 20 laments also that "the average physician is inclined to give no attention to" that "mental aspect" which "every illness" possesses.

For years past the present writer has been at pains to appreciate the significance of psychogenesis and the necessity for psychotherapy. Experience both in public hospital work and in private practice amply confirms T. A. Ross's teaching that it is often readily to be seen that a nervous illness confers some advantage on a patient. Until he (or she) sees that it is of greater advantage not to have it there will be a persistent, though unconscious, clinging to it. It pays to have a nervous illness, says Ross. Further, the process of securing the patient's enlightenment is often of long duration. It may not be hurried. It is time-consuming and brain-wearying for the practitioner, who gets very scant recognition for his efforts. Is it any wonder, then, that "the average physician" gives no attention to it? At this juncture may the question be put, in the changes in general practice now in contemplation for

Australia what are the inducements held out to influence the aforesaid "average physician" to give the desired attention to the mental aspect of disease?

May I ask also if it is not the experience of others besides myself that the worst people to treat are those whose treatment costs them nothing? Is not the necessity to pay a fee (even if small) a distinct help to the patient himself? Does it not make for his recovery from a psychogenic illness?

Is it really a matter of wonder that psychosomatic illness is increasing? Finally, is the manufacture of psychosomatic illnesses one of those secondary industries which call for Australian encouragement?

Yours, etc.,

R. C. EDWARDS.

Dulwich Hill,
New South Wales,
August 22, 1938.

SIR: Many have written and spoken for reduction of the income limit for contributors. All are in the wrong direction, for that but perpetuates our original error and admits the principle of the concession rate and service, and tends again to useless waste of calculation of lodge figures—an error perhaps to be repeated before the Royal Commission.

That basis is wrong, and if we must have this insurance there should be no income limit at all; then the capitation fee will be recognized as that which, applied universally, will yield an adequate remuneration and the principle be recognized of equal service for rich and poor.

Yours, etc.,

C. C. MCKELLAR.

North Bondi,
New South Wales,
August 25, 1938.

TOXIC GOITRE, WITH SPECIAL REFERENCE TO END-RESULTS.

SIR: The Lister orator (THE MEDICAL JOURNAL OF AUSTRALIA, August 20, 1938), in presenting the results of his experience in the treatment of toxic goitre, lays himself open to criticism when he voices the opinion that "X ray treatment is popular only in those places where surgical treatment is poor", and omits altogether reference to radium treatment. May it not be that the reverse is the case, and that surgical treatment is popular where radiation treatment is poor or its value unappreciated or ignored?

During the last ten years the writer has had experience of a goodly number of cases where operation for toxic goitre has been rendered unnecessary by the judicious use of X or radium rays upon the thyreoid during the period of rest and medication prior to proposed surgical removal by himself or other surgeons.

If team work upon toxic goitre is to be encouraged, surely it is necessary to remember that success in treatment by radiation is often obtainable, and that it will depend upon the recognition of the fact that the details of the techniques employed are just as important as the minutiæ of medicinal or operative measures.

Yours, etc.,

F. P. SANDES, F.R.A.C.S.,
Honorary Consulting Surgeon,
Royal Prince Alfred Hospital,
Sydney.

175, Macquarie Street,
Sydney,
August 24, 1938.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts, 1925 to 1935*, of Queensland, as duly qualified medical practitioners:

McGree, John Allan, M.B., B.S., 1933 (Univ. Sydney), Gladstone.

Nash, Eccles Norman, M.B., 1938 (Univ. Sydney), Brisbane.

Pottinger, George Wilfred, M.B., B.S., 1929 (Univ. Sydney), F.R.C.S. (Edinburgh), 1934, Sydney.

Wilkinson, Herbert John, M.B., Ch.M., 1925 (Univ. Sydney), M.D., 1930 (Univ. Sydney), M.D., 1934 (Univ. Adelaide), Brisbane.

VICTORIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1928, of Victoria, as a duly qualified medical practitioner:

Cohn, Ernst, L.R.C.P. *et* S. (Edinburgh), L.R.F.P.S. (Glasgow), 1938.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919, of South Australia, as duly qualified medical practitioners:

Phipps, Kathleen Elizabeth, M.B., B.S., 1922 (Univ. Melbourne), Morgan.

O'Loughlin, Irene May, M.B., B.S., 1938 (Univ. Adelaide), Adelaide.

Obituary.

CARL VIVIAN STEPHENS.

WE regret to announce the death of Dr. Carl Vivian Stephens, which occurred on August 22, 1938, at Melbourne, Victoria.

FREDERICK SHORT.

WE regret to announce the death of Dr. Frederick Short, which occurred recently at Epping, New South Wales.

WILFRED JAMES WHITE.

WE regret to announce the death of Dr. Wilfred James White, which occurred on August 27, 1938, at Sydney, New South Wales.

Books Received.

THE CONSTRUCTION OF VULCANITE APPLICATORS FOR APPLYING RADIUM TO LESIONS OF THE BUCCAL CAVITY, LIPS, ORBIT AND ANTRUM, by D. G. Walker, M.A., M.Dent.Sc., B.Ch., with a foreword by W. W. James, O.B.E., F.R.C.S., L.D.S.; 1938. London: John Murray, for the Middlesex Hospital Press. Demy 8vo, pp. 70, with 23 plates. Price: 7s. 6d. net.

A TEXTBOOK OF NEURO-RADIOLOGY, by C. P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S., and A. Orley, M.D., D.M.R.E.; 1938. London: Baillière, Tindall and Cox. Super royal 8vo, pp. 350, with illustrations. Price: 25s. net.

TREATMENT IN GENERAL PRACTICE (ARTICLES REPUBLISHED FROM THE BRITISH MEDICAL JOURNAL): THE MANAGEMENT OF SOME MAJOR MEDICAL DISORDERS; Second Edition; 1938. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 264. Price: 5s. 6d. net.

A GUIDE TO ANATOMY FOR STUDENTS OF MEDICAL GYMNASTICS, MASSAGE, AND MEDICAL ELECTRICITY, by E. D. Ewart; Fourth Edition; 1938. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 354, with 102 illustrations, including 39 plates. Price: 12s. 6d. net.

ARTHRITIS, FIBROSITIS AND GOUT: A HANDBOOK FOR THE GENERAL PRACTITIONER, by C. W. Buckley, M.D., F.R.C.P.; 1938. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 162, with 14 plates. Price: 7s. 6d. net.

SULFANILAMIDE THERAPY OF BACTERIAL INFECTIONS, WITH SPECIAL REFERENCE TO DISEASES CAUSED BY HEMOLYTIC STREPTOCOCCI, PNEUMOCOCCI, MENINGOCOCCI AND GONOCOCCI, by R. R. Mellon, M.D., D.P.H., D.Sc., P. Gross, M.D., and F. B. Cooper, M.S.; 1938. Baltimore: C. C. Thomas. Medium 8vo, pp. 409. Price: $4.00 net.

A GUIDE TO HUMAN PARASITOLOGY FOR MEDICAL PRACTITIONERS, by D. B. Blacklock, M.D., D.P.H., D.T.M., and T. Southwell, D.Sc., Ph.D., A.R.C.Sc., F.Z.S., F.R.S.E.; Third Edition; 1938. London: H. K. Lewis and Company Limited. Royal 8vo, pp. 268, with 2 coloured plates and 122 illustrations in the text. Price: 12s. 6d. net.

AIDS TO PRACTICAL NURSING, by M. Houghton, S.R.N., S.C.M., D.N., with a foreword by M. L. Rosenheim, M.A., M.B., M.R.C.P.; 1938. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 320, with illustrations. Price: 3s. 6d. net.

AIDS TO HYGIENE FOR NURSES, by E. M. Funnell, S.R.N., D.N., with a foreword by C. Muir, M.B., Ch.B., D.P.H.; 1938. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 235, with illustrations. Price: 3s. 6d. net.

AIDS TO SURGICAL NURSING, by K. F. Armstrong, S.R.N., S.C.M., with a foreword by C. P. G. Wakeley, D.Sc., F.R.C.S.; 1938. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 319, with illustrations. Price: 3s. 6d. net.

REPORTS ON CHRONIC RHEUMATIC DISEASES, BEING THE ANNUAL REPORT OF THE BRITISH COMMITTEE ON CHRONIC RHEUMATIC DISEASES APPOINTED BY THE ROYAL COLLEGE OF PHYSICIANS: Number IV, edited by C. W. Buckley, M.D., F.R.C.P.; 1938. London: H. K. Lewis and Company Limited. Royal 8vo, pp. 172, with five plates. Price: 10s. 6d. net.

Diary for the Month.

SEPT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 7.—Victorian Branch, B.M.A.: Branch.
SEPT. 9.—Queensland Branch, B.M.A.: Council.
SEPT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 20.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: Branch.
SEPT. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
SEPT. 23.—Queensland Branch, B.M.A.: Council.
SEPT. 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Victorian Branch, B.M.A.: Council.
SEPT. 29.—South Australian Branch, B.M.A.: Branch.
SEPT. 29.—New South Wales Branch, B.M.A.: Branch.
SEPT. 30.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages XX to XXII.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Medical Staff.
THE BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, NEW SOUTH WALES: Acting Assistant Surgeon
THE QUEEN'S (MATERNITY) HOME INCORPORATED, ADELAIDE, SOUTH AUSTRALIA: Resident House Surgeon.
VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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